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CONTENTS OF PREVIOUS NUMBERS

OCTOBER, 1920. NUMBER 4

NOVEMBER, 1920. NUMBER 5

DECEMBER 1920, NUMBER 6

CONTENTS OF VOLUME 5

JANUARY, 1921. NUMBER 1

FEBRUARY, 1921. NUMBER 2

MARCH, 1921. NUMBER 3

APRIL, 1921. NUMBER 4

MAY, 1921. NUMBER 5

JUNE, 1921. NUMBER 6

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A CLINICAL AND ANATOMIC STUDY OF A VASCULAR LESION OF BOTH CEREBELLAR HEMISPHERES

WITH ESPECIAL REFERENCE TO CEREBELLAR CATALEPSY AND NYSTAGMUS, AND THE ANATOMIC CONNECTIONS OF THE INFERIOR OLIVE *

WALTER F. SCHALLER, M.D.
Associate Professor of Medicine (Neurology), Leland Stanford Junior University
SAN FRANCISCO

CLINICAL STUDY

History.—J. E. S., aged 58, a native of Boston, an inmate of the Relief Home, San Francisco, was first examined by me on Dec. 7, 1911. His illness began with a sudden onset three years before, in Japan, without previous ill health or warning. Seated in a tea-house in Yokohama, he experienced a sensation as if he were "struck by a ball of lightning in the knees." He arose and attempted to walk, which he was able to do for a distance of about 25 yards, but would then have fallen had he not been aided. There was no loss of consciousness, no pain, headache, nausea or vomiting. Incontinence of urine immediately followed the attack, and later there was imperative micturition. Following the stroke he remained in bed several days. It was at once apparent that control of his left arm and leg was practically lost for all voluntary movements. The right arm and leg were also affected but to a lesser extent. Being right-handed, he could feed himself but with considerable difficulty. It was not possible for him to walk or even to stand without support, and he would sit all day in a wheel chair. His speech had changed, having become slow and deliberate without any disturbance of pronunciation. Later, hearing and vision were affected; vision was affected for near objects but not for distance. The patient believed that his mentality was as good as formerly, that his memory was as good, and that his disposition had not changed. He was not emotional. There had been no remarkable change in his condition in the three years that had elapsed. He did not complain of weakness, and with the exception of his great loss of muscular control and imperative micturition, he considered himself in average health.

The family and previous personal history of the patient were unimportant. Well as a child, his first serious illness was an attack of typhoid in 1898. Venereal infection was denied. He was a total abstainer from alcohol, but smoked in moderation.

Examination.—Examination revealed a man apparently of the given age, practically helpless because of great motor incoordination in all extremities

* Read at the Forty-Sixth Annual Meeting of the American Neurological Association, New York, June, 1920.

* From the Neurological Clinic, Department of Medicine, Leland Stanford Junior University, San Francisco.

but especially in the left extremities. To walk was impossible and even to attempt it was to risk a fall. The patient could scarcely stand by supporting himself with both hands on the bed rail. He would sit upright in his wheel chair for hours at a time with no discomfort or difficulty, and he spent his days in this manner. Intelligence and emotions were evidently not impaired. Speech was typically scanning, but there was no dysarthria.

An analysis of his disturbed coordination was attempted. In the different tests the incoordination was of the same character on both sides but far greater on the left. When the patient was directed to place his index finger on the tip of his nose he would always pass it, sometimes moving his hand several times back and forth in a jerky awkward fashion, giving the impression that the orientation was unimpaired. This was supported by the fact that the same errors were made with the eyes open or shut. The same phenomena was found in the heel to knee test. This failure of attaining the designated object was interpreted as a mouvement démesuré (Thomas and Jumentié) and was probably dependent on disturbed muscle synergy.

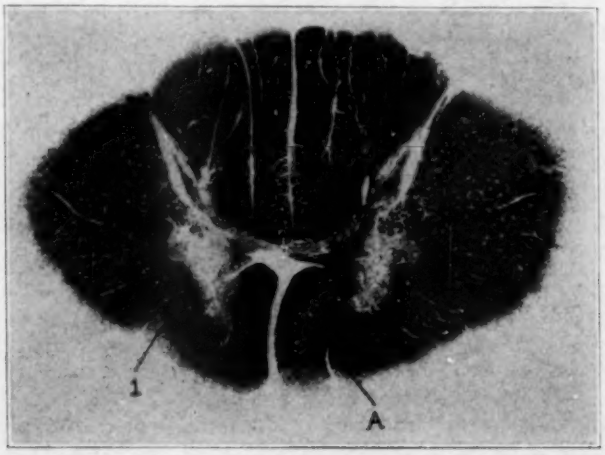

Fig. 1.—Mid-Cervical cord: 1, degeneration of left Helweg's bundle; A, degeneration in the right anterior column. In this and in the following illustrations the left side of the figures corresponds to the left side of the brain.

Disturbance of synergy in this patient was examined by Babinski tests, which would tend to dissociate muscular movements. When the patient in the sitting position was directed to touch with his foot the examiner's hand held at about the level of the trunk in front of him, there would at first be a flexion of the thigh on the pelvis and then an extension of the leg, the foot finally attaining the mark in this fashion. In the upper extremities the alternate supination and pronation of the hand revealed great deficiency in the rapid and accurate successive contractions of antagonistic muscles; there were adiadokokinesis in the left hand, causing wide excursion of the forearm, and dysdiadokokinesia in the right.

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1. Babinski: De l'équilibre volitionnel statique et de l'équilibre volitionnel cinetique, Rev. Neurol. **10**:470, 1902.

Perhaps the most striking symptom in this case was that of cerebellar catalepsy (Babinski). When the patient lay on his back the thighs flexed on the pelvis and somewhat abducted and the legs on the thighs, after a few unsteady movements of the extremities, a remarkable immobility ensued. Not only was this immobile posture noteworthy in one who showed such great disturbance of voluntary motion, but it was actually more pronounced than in the normal case, and the duration was longer. This symptom was tested repeatedly and found to be constant.

It was not noted that the patient had a tendency to fall in any one direction when he lost his equilibrium, that there was hypotonia present or tendency to fixed attitudes. The muscular force of the extremities was well preserved.

Voltaic vertigo was tested from the standpoint of the functional labyrinth tests. With an interrupted current of from 14 to 16 ma., with both electrodes above and in front of the tragus, a slight inclination of the head was produced toward the side of the positive pole. Turning tests or syringing were

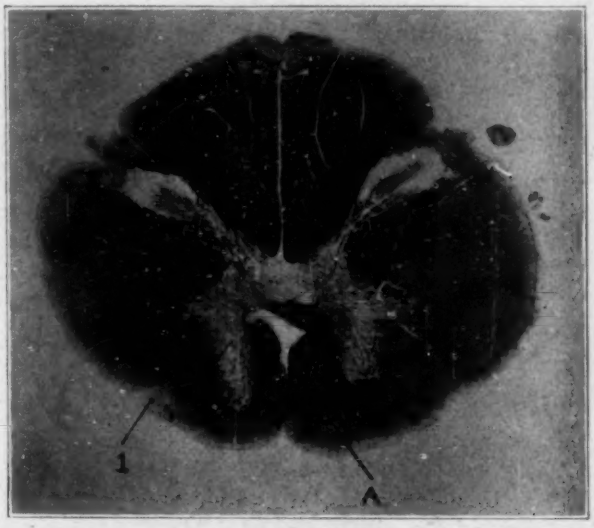

Fig. 2.—Upper cervical cord: 1, degeneration of left Helweg's bundle; A, degeneration in right anterior column.

not performed. Spontaneous past pointing after Bárány was tested, but pointing was always done correctly in the sense that allowance being made for dyssynergia, there was no constant tendency to deviate to one side. It is not noted with which portion of the extremities these tests were made.

In the general neurologic examination there were no muscular atrophy, tremor or fibrillations present. It was noted that the left tendon reflexes were increased over those on the right, with the exception of the Achilles' reflexes, which were equal. The Babinski reflex was absent. Sensibility tests for light touch, pain and temperature were normal throughout. The stereognostic sense was intact. Postural sense and osseous sensibility were not impaired.

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2. Babinski: Proceedings XVIIth International Congress of Medicine, London, 1913; Section XI, Neuropathology.

Cranial Nerves: The pupils were about equal in size but somewhat irregular in outline and reacted to light, accommodation and convergence. There was no spontaneous nystagmus in looking forward to the extreme right or left, or upward. The movements of the eyeballs were normal, but it seemed to be an effort for the patient to look upward, and he complained of pain in the muscles at the back of the neck when he did so. The corneal reflex was present. Fields of vision were normal by roughly testing. Sensation over the face was normal. Hearing was diminished in both ears to watch tick. There was no facial paralysis. The walls of the soft palate moved equally and well, and the pharyngeal reflex was active. There were no signs of bulbar paralysis. The spinal accessory nerve was normal. The tongue protruded in the mid-line without tremor.

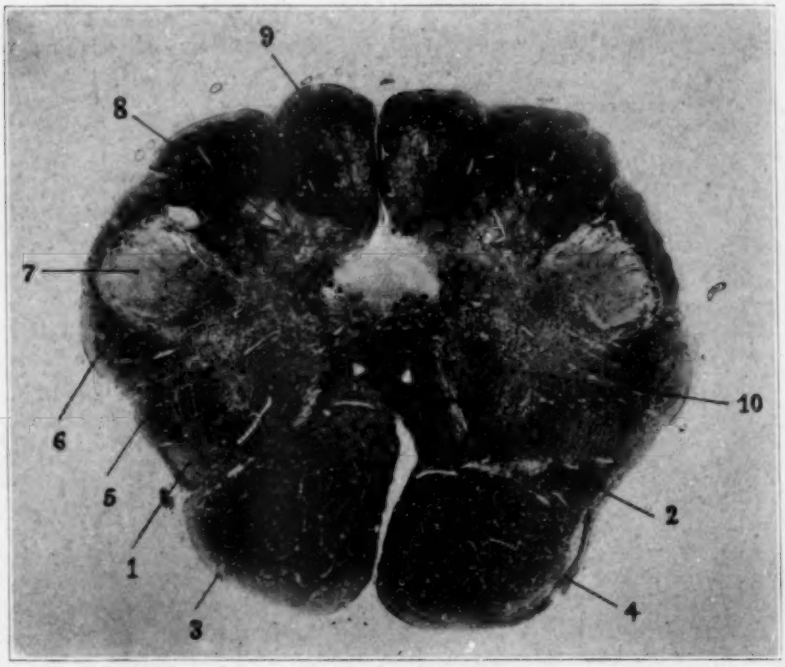

Fig. 3.—Transverse section of medulla at the beginning of the pyramidal decussation: 1, degeneration of left Helweg's bundle; 2, right Helweg's bundle; 3, degeneration of left lateral external arcuate fibers; 4, right lateral external arcuate fibers; 5, Gower's tract; 6, direct cerebellar tract; 7, substantia gelatinosa; 8, nucleus cuneatus; 9, nucleus gracilis; 10, pyramidal decussation.

The patient was well nourished, ate and slept well. The cardiovascular system seemed to function well. The pulse was of fair tension and volume, and the radial artery was not appreciably hardened. The heart was not enlarged, and no bruits were heard. The abdomen and lungs were reported normal, with the exception of a slight emphysema.

Because of the history of sudden onset, the stationary character of the affection and the characteristic cerebellar symptomatology which was predominant on the left side, a diagnosis of a vascular lesion in the left cere-

bellar hemisphere was made. The patient died suddenly on Feb. 15, 1912, and it was suspected that he suffered an apoplectic stroke.

ANATOMIC STUDY

The Lesion.—The brain and cord alone were removed at necropsy so that the exact cause of death was not determined. A marked arteriosclerosis, particularly at the base of the brain, was observed, but no thickening of membranes, adhesions or tumor mass was found. External to the left superior peduncle (Fig. 11, No. 58), a small area of superficial softening was seen; otherwise the external appearance of the brain was normal. The brain was hardened in a 10 per cent. solu-

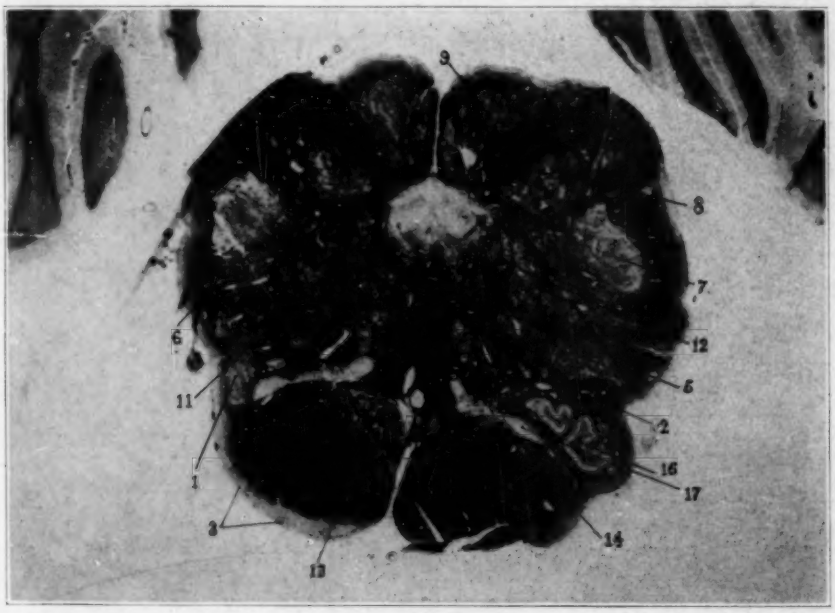

Fig. 4.—Transverse section at caudal extremity of inferior olive: 1, degeneration of left Helweg's bundle; 2, right Helweg's bundle; 3, degeneration of left lateral external arcuate fibers; 5, Gower's tract; 6, direct cerebellar tract; 7, substantia gelatinosa; 8, nucleus cuneatus; 9, nucleus gracilis; 11, left internal arcuate fibers; 12, degeneration of right internal arcuate fibers; 13, degeneration of left median fillet; 14, right median fillet; 16, right inferior olive; 17, medial accessory olive.

tion of formaldehyd before it was cut. It was then studied macroscopically by serial sections; the cerebellum and medulla were removed by a transverse cut through the peduncles, and the hemispheres were divided by cutting through the corpus callosum. Both cerebral hemispheres were then cut horizontally into a number of slices, after the procedure of Marie. No lesions were found. Transverse sections through

the cerebellum, however, revealed old symmetrical areas of softening in the central white of both hemispheres with cavity formation, the destruction of tissue being greater on the left side. In neither hemisphere did the softening reach the periphery. In both hemispheres the lesion extended well backward toward the posterior poles; on the left side (Figs. 8, 9 and 10, Nos. 27 and 28) it destroyed the dentate nucleus, nucleus emboliformus and globosus, and extended well forward toward the anterior pole, as seen by fiber degeneration dorsal and lateral to the superior peduncle (Fig. 11, No. 58). In the right hemisphere the dentate nucleus and accessory nuclei were conserved, but laterally and

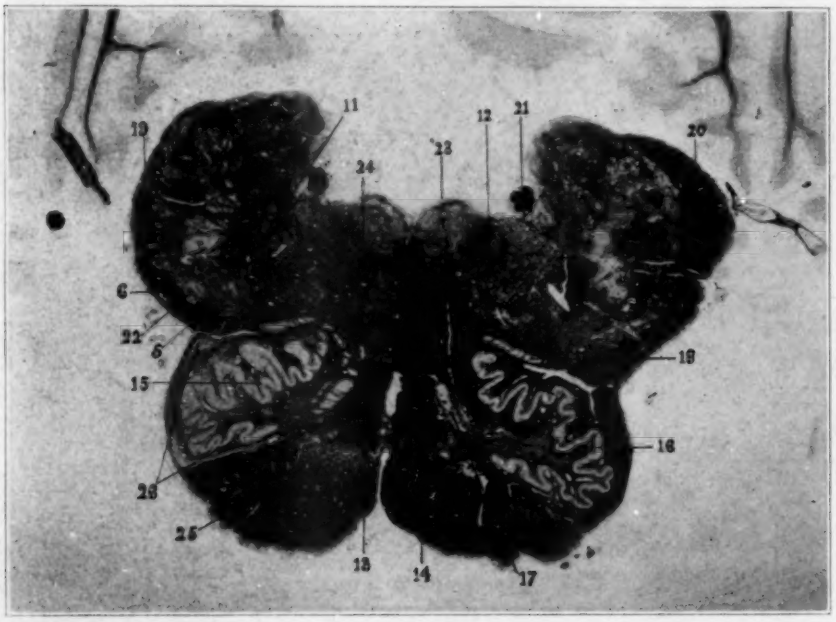

Fig. 5.—Transverse section through lower olive: 5, Gower's tract; 6, direct cerebellar tract; 11, left internal arcuate fibers; 12, degeneration of right internal arcuate fibers; 13, degeneration of left median fillet; 14, right median fillet; 15, left inferior olive; 16, right inferior olive; 17, medial accessory olive; 18, dorsal accessory olive; 19, left restiform body; 20, right restiform body; 21, fasciculus solitarius; 22, lateral nucleus; 23, hypoglossal nucleus; 24, root fibers of hypoglossal nerve; 25, degeneration in left pyramid; 26, peri-olivary degeneration.

below there were seen degenerations which involved the middle cerebellar peduncle. The vermis and roof nuclei were not involved in the destructive process, and no degenerations were present.

In addition to these large lesions, there were three other minor lesions, one mentioned above found external to the left superior peduncle, and another (Fig. 9, No. 49) central and found at its greatest

extent below the sixth nerve nucleus on the left side. The latter reached to the midline, not measuring over 1.5 mm. at its greatest transverse diameter. It did not extend below the upper level of the inferior olive, and traced upward it was seen to disappear at the crossing of the trapezoid body, a number of whose fibers it divided. The third small lesion (Fig. 12, No. 68), a degeneration in the right crusta, was lost in lower sections and traced upward finally disappeared at the level of the upper end of the red nucleus. This degeneration was due to a local softening from thrombosis of smaller vessels.

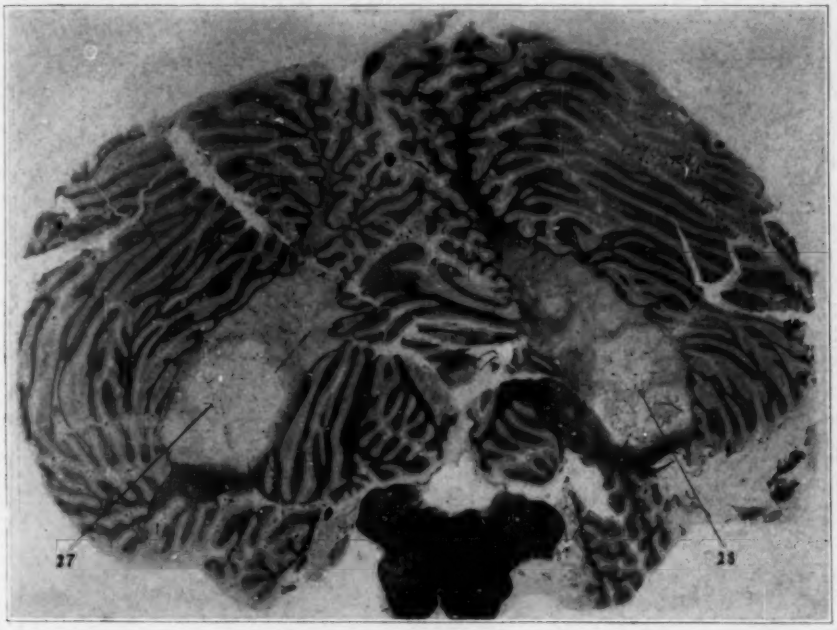

Fig. 6.—Transverse section of posterior cerebellum and medulla: 27, softening in left cerebellar hemisphere; 28, softening in right cerebellar hemisphere.

Fixation and Staining.—The entire brain and cord were hardened in bichromate solution. The rhombencephalon and portions of the upper cervical and dorsal and lumbar cords were included in celloidin, blocked and cut in serial transverse sections and stained by the Kulschitzky and Weigert methods, alternate sections being counterstained by cochineal. Subsequently, the peduncular and subthalamic regions were reconstructed in one block, and the basal ganglions in another, so that eventually three separate blocks were serially sectioned, including all parts of the brain except the upper cortex, frontal and occipital poles. Due to the extended study of these different blocks and the interruption occasioned by the World War, the final report of this

case was delayed until the present time. Mention is made of this case, however, in the *California State Journal* for July, 1913, and in the *Journal of Nervous and Mental Diseases* for May, 1915.

The Degenerations.—Striking degenerations occasioned by the cerebellar lesions were the practically complete degeneration of the left superior peduncle, degeneration of the right red nucleus and in Forel's field, degeneration of the left restiform body and atrophy of the right inferior olive. The atrophy of the right olive was evidenced by its small size, principally due to diminution in width of the cellular layer and its poverty of cells. However, both the internal fibers entering the

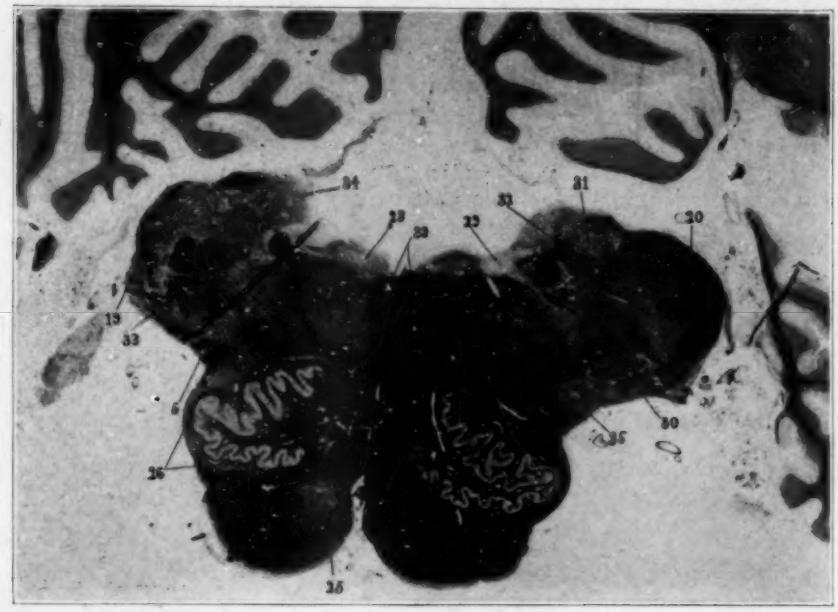

Fig. 7.—Enlargement of medulla in Figure 6. Section at emergence of vagus nerve: 5, Gower's tract; 19, left restiform body; 20, right restiform body; 23, hypoglossal nucleus; 25, degeneration in left pyramid; 26, peri-olivary degeneration; 29, dorsal vagus nucleus; 30, root fibers of vagus nerve; 31, Deiters' nucleus; 32, descending vestibular root; 33, spinal root of fifth nerve; 34, median vestibular nucleus; 35, olivocerebellar fibers; 39, posterior longitudinal fasciculi.

hilum and those traversing the gray substance to attain the periphery were well preserved. The left olive was not without alteration. This was due to a marked fiber decrease in its outer half shown in the strands traversing the gray substance and evidently continuous with the peri-olivary degeneration presently to be described. The fine fiber felt-work between the cells in the gray substance was also much less marked than in the opposite or degenerated right olive. The gray matter,

however, was apparently well conserved. The cellular changes in the right medial and right dorsal accessory olives were similar to those found in the right inferior olive. The left olivocerebellar fibers were degenerated (Fig. 7). The left nucleus lateralis was, possibly, poorer in cells than the right. The left olive was brought out in relief by a degeneration of fibers lateral and ventral to it—peri-olivary fibers (Figs. 5 and 6, No. 26). A degeneration was traced cephalad as well as caudad in relation to the olive, and it seemed to be continuous. This will be discussed later. The median fillet was less darkly stained on

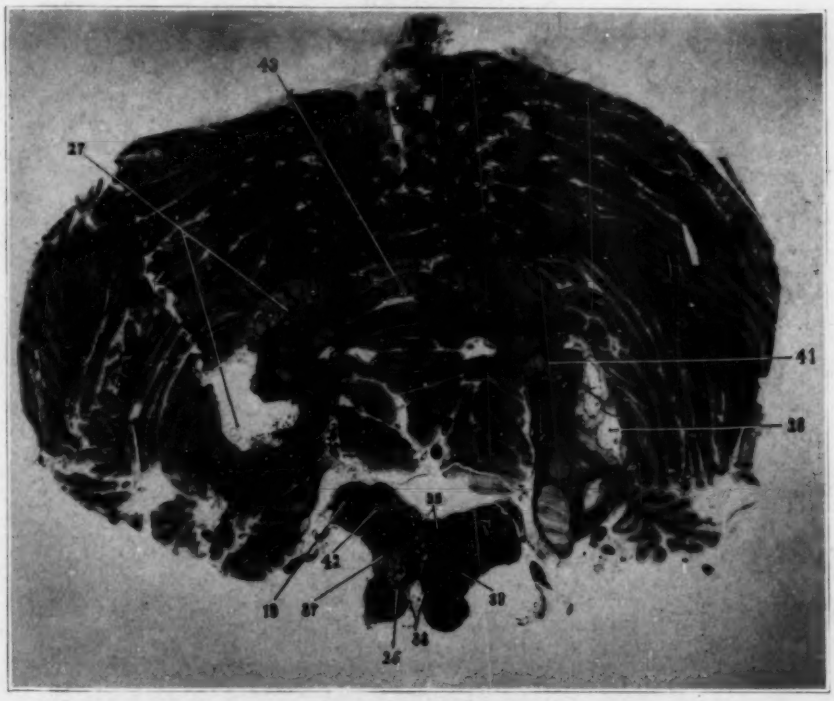

Fig. 8.—Transverse section of cerebellum and of medulla at emergence of glossopharyngeal nerve: 19, left restiform body; 25, degeneration in left pyramid; 27, softening in left cerebellar hemisphere; 28, softening in right cerebellar hemisphere; 36, arcuate nuclei; 37, degeneration of left central tegmental tract; 38, right central tegmental tract; 41, right dentate nucleus; 42, root fibers of glossopharyngeal nerve; 43, vermis.

the left side, and the crossing right internal arcuate fibers to it from the right posterior column nuclei were greatly diminished in number (Fig. 4, No. 12). The right nucleus cuneatus was poor in cells. The left anterior and anterolateral external arcuate fibers were degenerated (Figs. 3 and 4, No. 3).

In the left pyramid at its median and dorsal aspect there was a degeneration of fibers which was quite distinct in the region of the olive (Figs. 5, 6 and 7, No. 25) but it became much less so at the pyramidal decussation and was not seen in the cervical cord. Upward this degeneration was lost in the pyramidal bundles traversing the pons. In its greatest extent the degeneration seemed to radiate from the left arcuate nucleus. The arcuate nuclei, except in their lower portions, stained poorly with cochineal. There was no remarkable difference in their appearance.

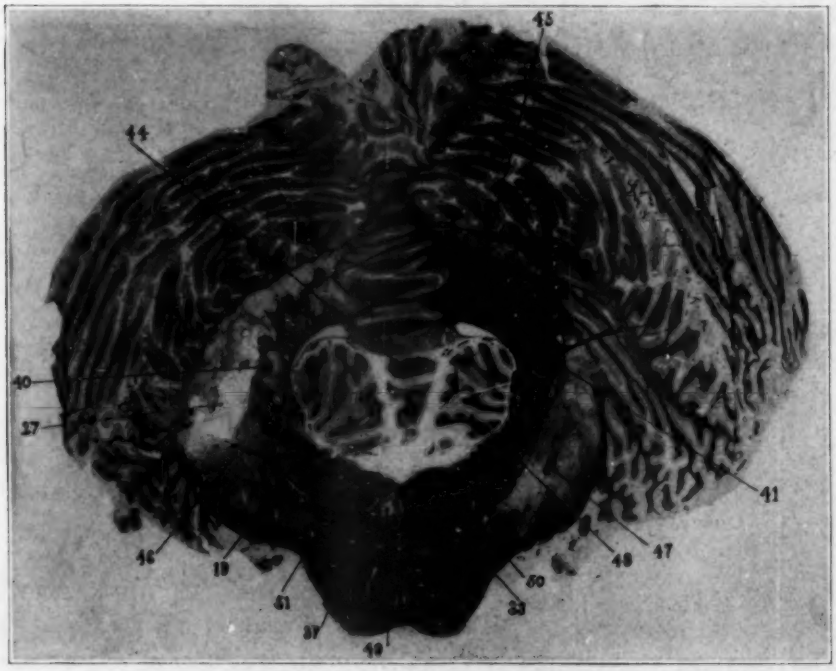

Fig. 9.—Transverse section of medulla and of pons through nucleus of sixth nerve: 19, left restiform body; 27, softening in left cerebellar hemisphere; 37, degeneration of left central tegmental tract; 38, right central tegmental tract; 40, degeneration of left dentate nucleus; 41, right dentate nucleus; 44, inferior vermis; 45, nucleus globosus; 46, left middle cerebellar peduncle; 47, right middle cerebellar peduncle; 48, tonsil; 49, vascular lesion in left median fillet; 50, abducens nucleus; 51, trapezoidal fibers.

The middle peduncles were severely degenerated on both sides (Figs. 9 and 10, Nos. 46 and 47), and the transverse fibers of the pons were degenerated (Fig. 11, No. 61). This was also true of the ascending pons fibers to the tegmentum. In contrast to the fiber degeneration, the pontile nuclei were remarkably conserved. In the tegmentum the left median fillet was seen to be involved by the small central lesion mentioned above (Fig. 9, 49). Some of the fibers of the

left posterior longitudinal bundle were involved. Also a degeneration of the left central tegmental tract was present over the normal left olive (Figs. 8, 9, 10 and 11, No. 37). The corpora quadrigemina appeared to be normal.

On the left side the cells of Deiter's nucleus might be somewhat diminished in number, as well as the fibers of the descending vestibular root (juxta restiform body. On the same side, in higher sections, con-

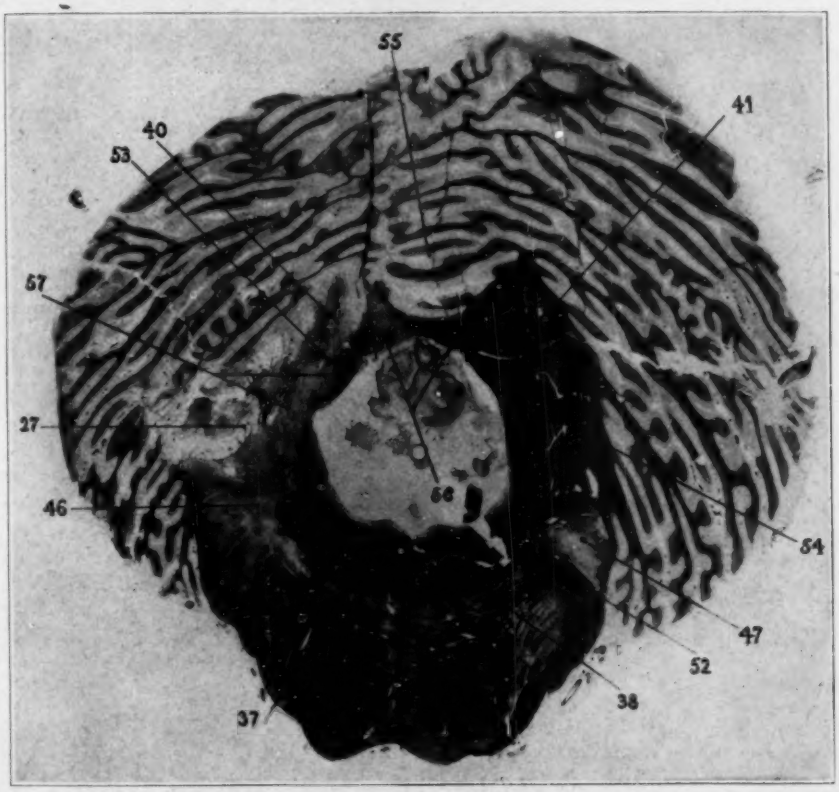

Fig. 10.—Transverse section of cerebellum and of pons at emergence of fifth nerve: 27, softening in left cerebellar hemisphere; 37, degeneration of left central tegmental tract; 38, right central tegmental tract; 40, degeneration of left dentate nucleus; 41, right dentate nucleus; 46, left middle cerebellar peduncle; 47, right middle cerebellar peduncle; 52, fibers of fifth nerve; 53, degeneration of left superior cerebellar peduncle; 54, right superior peduncle; 55, superior vermis; 56, roof nuclei; 57, fibers between tegmentum pontis and roof nuclei.

served strands were seen which extended between the tegmentum pontis and the roof nuclei (Fig. 10, No. 57). There was a lateral portion which lay lateral to the degenerated superior peduncle and a medial por-

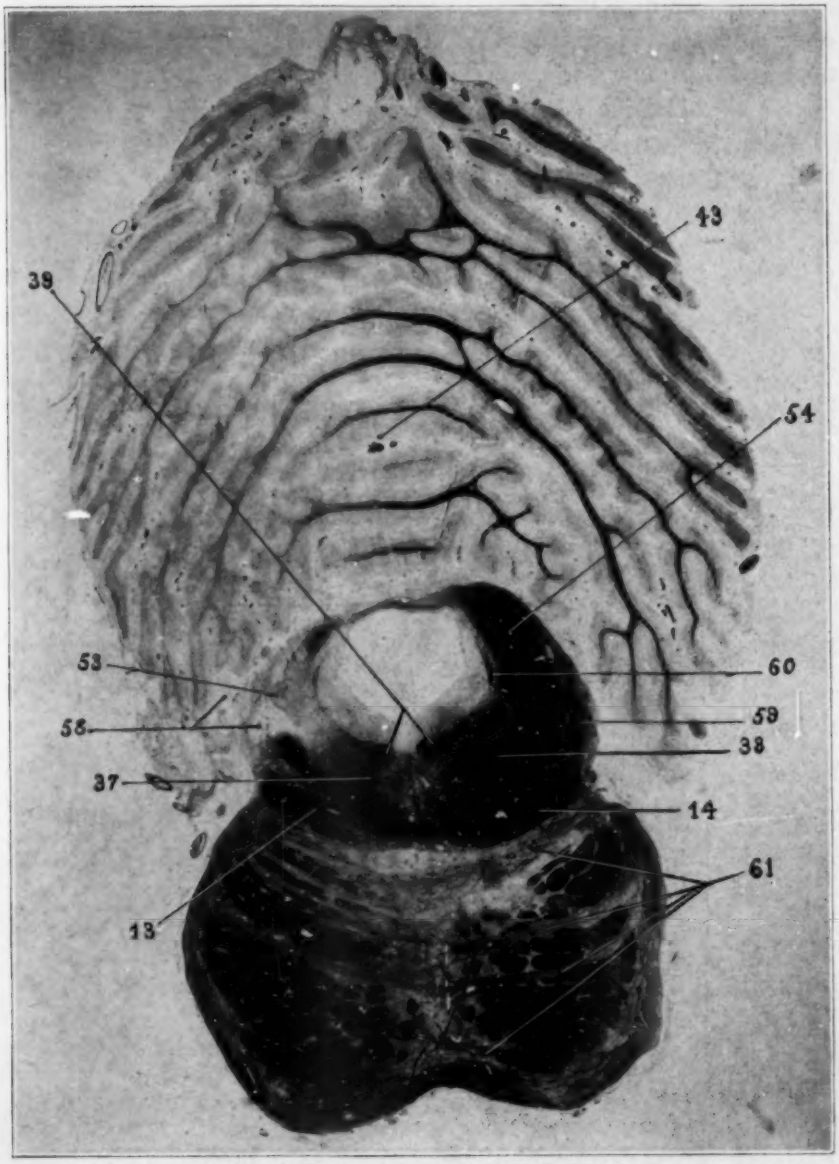

Fig. 11.—Transverse section through the isthmus: 13, degeneration of left median fillet; 14, right median fillet; 37, degeneration of left tegmental tract; 38, right central tegmental tract; 39, posterior longitudinal fasciculi; 43, vermis; 53, degeneration of left cerebellar peduncle; 54, right cerebellar peduncle; 58, vascular lesion involving left lateral fillet; 59, right lateral fillet; 60, mesencephalic root of trigeminus; 61, degeneration of transverse pontile fibers.

tion bordering the ventricular wall. Fibers in the decussation of the roof nuclei were continuous with the foregoing in a sweeping curve. It is probable that these fibers represented the nucleocerebellar tract or the fastigiobulbar tract, or both. The right roof nucleus was intact, the left relatively little less poor in number of cells or staining qualities.

Because of the changed direction of the cuts from transverse to horizontal in the second block, which included the peduncular and subthalamic regions, some of the lower sections were incomplete. Also, in the reconstruction of the specimens, the right half of these sections was misplaced posteriorly and slightly downward.

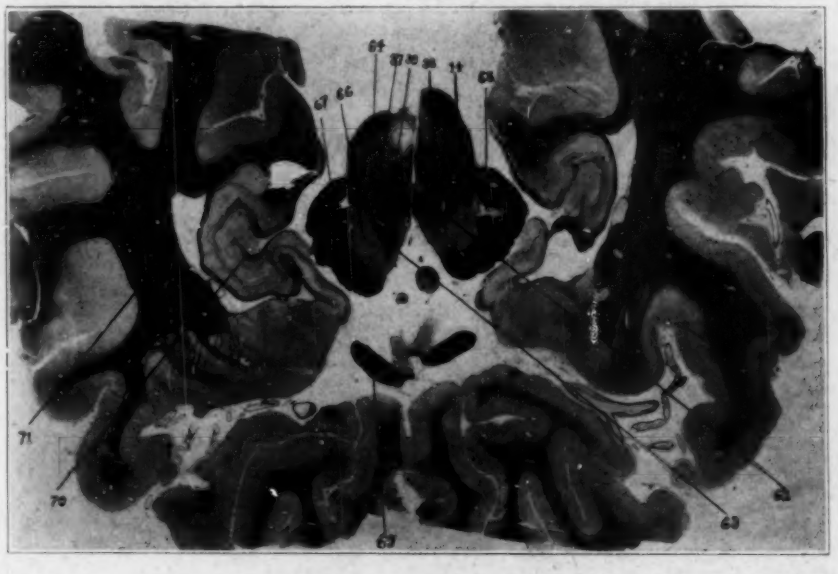

Fig. 12.—Horizontal section through red nucleus. The right half of the section and following sections are misplaced posteriorly and slightly downward because of the reconstruction of the specimen: 14, right median fillet; 37, degeneration of left central tegmental tract; 38, right central tegmental tract; 39, posterior longitudinal fasciculus and nucleus of third nerve; 62, degenerated right red nucleus; 63, root fibers of oculomotor nerve; 64, inferior colliculus; 66, substantia nigra; 67, pes pedunculi; 68, vascular lesion in right pes pedunculi; 69, optic tract; 70, hippocampus; 71, inferior longitudinal fasciculus.

The right red nucleus (Figs. 12 and 13, No. 62) showed a marked degeneration as compared with the left side, shown by the poverty of transversely cut fibers. Above the degenerated red nucleus in the subthalamic region (Fig. 14, No. 78) there was a considerable thinning of fibers in Forel's field, notably in the thalamic bundle of Forel (Déjerine) (Figs. 15 and 16, No. 88). Yet these fibers seemed to be distinct from a bundle which was in relation to the mammillothal-

amic tract, and also designed by this author as part of the thalamic bundle. The lenticular bundle of Forel, ansa lenticularis, and inferior thalamic peduncle were unaffected (Fig. 15). In the optic thalamus, neither in the external nor internal medullary laminae or in the external nucleus of this body, could degenerations actually be demonstrated. For this reason, sections of the basal ganglions are not shown in the illustrations (Block 3). It is quite remarkable that the demonstrable degenerations above the red nucleus were so slight. This would suggest considerable autonomy of the cerebellum and this nucleus.

In sections below the olive, replacing the peri-olivary degeneration on the left, there was a degeneration of fibers extending caudalward

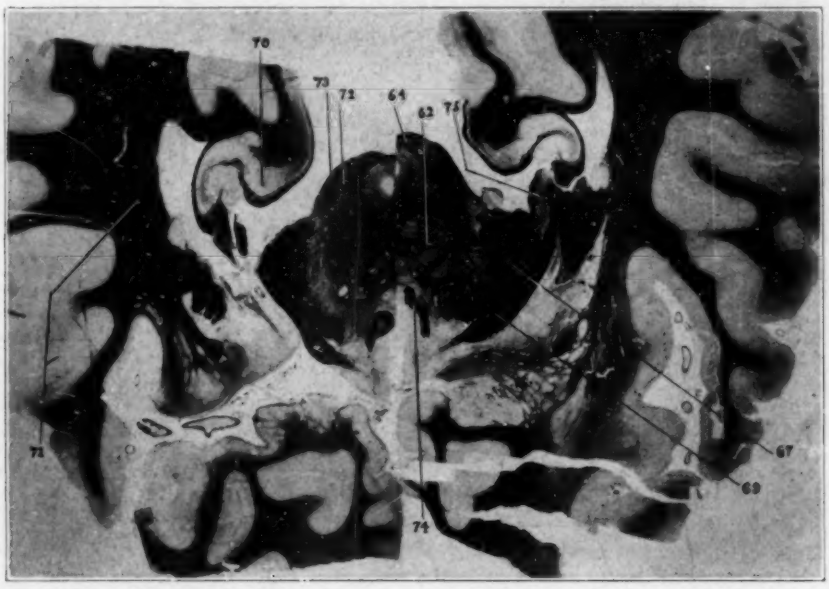

Fig. 13.—Horizontal section through optic tract: 62, degenerated right red nucleus; 64, inferior colliculus; 67, pes pedunculi; 69, optic tract; 70, hippocampus; 71, inferior longitudinal fasciculus; 72, inferior brachium; 73, superior brachium; 74, anterior pillar of fornix; 75, external geniculate body.

in the anterolateral bundle of fibers to the cord in the angle between the pyramid and the afferent cerebellar cord tracts (Fig. 3, No. 1). In the upper cervical cord it was traced as a triangular degeneration with its base at the periphery in the lateral column (Fig. 2); in the middle cervical region (Fig. 1) it was very much less marked and superficial, and it could not be found in the cervical enlargement. This degenerated fiber bundle was without doubt Helweg's tract. In the anterior column on the opposite or right side there was a slight thinning of fibers lateral to the direct pyramidal tract (Figs. 1 and 2 A).

It could be traced through the cervical cord and even in diminishing volume in the dorsal cord, but could not be seen in the lumbar region. This degeneration probably marked the descending cerebellospinal tract. The spinocerebellar tracts were not degenerated.

INTERPRETATION AND DISCUSSION

The foregoing is the report of a case in which there is a destruction of tissue in both cerebellar hemispheres with conservation of the vermis and the central vestibular system, and whose predominant symptomatology is marked dyssynergia, cerebellar catalepsy and scanning speech. Equally important from the standpoint of negative symptomatology is

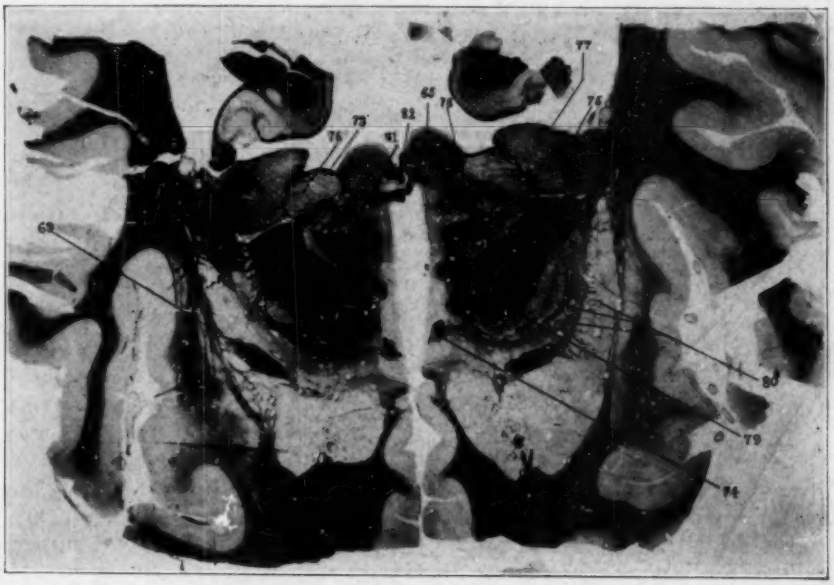

Fig. 14.—Subthalamic region. Horizontal section through superior colliculus: 65, superior colliculus; 69, optic tract; 73, superior brachium; 74, anterior pillar of fornix; 75, external geniculate body; 76, internal geniculate body; 77, pulvinar; 78, degeneration in right Forel's field; 79, mammillothalamic tract (Vicq d'Azyr); 80, lenticular nucleus; 81, Luys' body; 82, posterior commissure.

the absence of spontaneous nystagmus and of spontaneous errors of pointing after Bárány.

Cerebellar catalepsy was first described by Babinski [1] in a patient who presented, as in the case reported, a dissociation of the two kinds of volitional equilibrium: asynergia and catalepsy. He considered the latter as a symptom of cerebellar disease and, in discussing the underlying pathology quotes Dupré and Devaux, and Léopold Lévi as observing similar states in cerebellar abscess. In a later communica-

tion [2] Borgherini and Gallerani are quoted as observing a similar condition in animal experimentation on the cerebellum; and Rossi, in a case of parenchymatous atrophy of the cerebellum, noticed that this symptom was quite marked. Recently, La Salle Archambault [3] observed a similar case in which the degeneration of the Purkinje cells was the predominating and only constant feature. The central nuclei, olivary bodies and olivary cerebellar system, showed only insignificant secondary retrograde atrophies.

Déjerine [4] has stated that this symptom lacks anatomic confirmation as being due exclusively to a cerebellar lesion. Oppenheim [5] considered

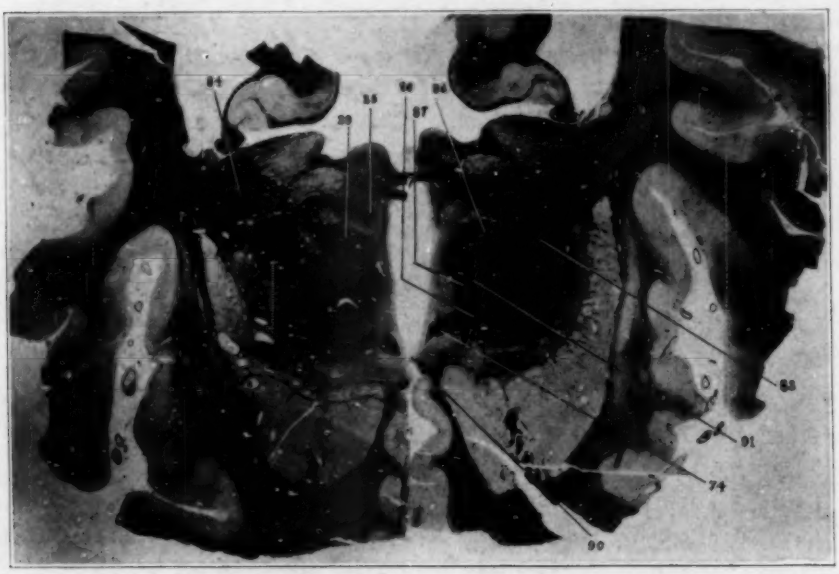

Fig. 15.—Subthalamic region. Horizontal section superior to Figure 13: 74, anterior pillar of fornix; 83, internal capsule; 84, Wernicke's field; 85, fasciculus retroflexus (Meynert); 86, ansa lenticularis; 87, inferior peduncle of thalamus; 88, degeneration in right thalamic bundle of Forel; 89, left thalamic bundle of Forel; 90, posterior commissure; 91, lenticular bundle of Forel.

this symptom quite rare and had not encountered it in his experience up to the year 1913.

The absence of nystagmus is explained by the practically intact vestibular system. This case confirms the opinion of Wilson and Pike [6]

3. Archambault, LaSalle: Parenchymatous Atrophy of the Cerebellum, J. Nerv. & Ment. Dis. **48**:273, 1918.

4. Déjerine: Semiologie System Nerveux **1**:424, 1914.

5. Oppenheim: Lehrbuch der Nervenkrankheiten **2**:1380, 1913.

6. Wilson, J. G., and Pike, F. H.: The Differential Diagnosis of Lesions of the Labyrinth and of the Cerebellum, J. A. M. A. **65**:2156 (Dec. 18) 1915.

that rhythmic nystagmus or labyrinthine nystagmus consisting of slow and quick components is properly a symptom of vestibular disease. They distinguish between this form and cerebellar nystagmus, characterizing the latter as an asynergia of ocular muscles of oscillatory nature. In Archambault's recent case of cerebellar atrophy with intact vestibular system, he speaks of "nystagmic shocks" rather than true nystagmus interpreted either as a tremor of the ocular muscles or perhaps as a manifestation of asynergy. Oppenheim, admitting that nystagmus is frequently given in the symptomatology of cerebellar lesions, doubts

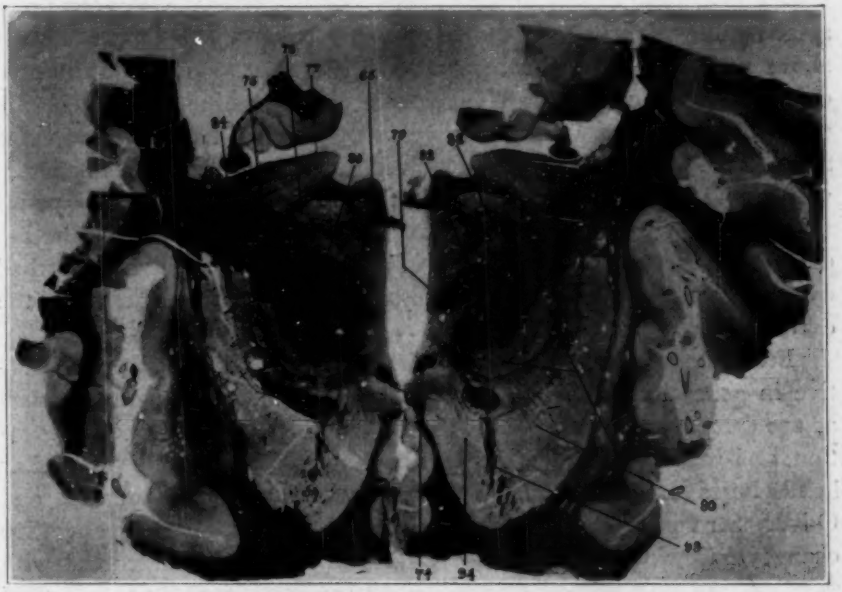

Fig. 16.—Subthalamic region. Horizontal section superior to Figure 14, passing through semilunar nucleus: 65, superior colliculus; 74, anterior pillar of fornix; 75, external geniculate body; 76, internal geniculate body; 77, pulvinar; 79, mammillothalamic tract (Vicq d'Azyr); 80, lenticular nucleus; 84, Wernicke's field; 88, degeneration in right thalamic bundle of Forel; 89, left thalamic bundle of Forel; 92, semilunar nucleus of Flechsig; 93, anterior segment of internal capsule; 94, caudate nucleus.

whether it may be due directly to cerebellar lesions. In 1915, I[7] expressed the opinion that nystagmus properly speaking is not a symptom of disturbed cerebellar function.

The normal outcome of pointing tests, although incomplete and only tested by spontaneous pointing and not after turning or syringing, are

7. Schaller: Cerebellar Syndrome, J. Nerv. & Ment. Dis. 42:270, 1915.

significant. From the standpoint of cerebellar function, the discussion relevant to the above may be best taken verbatim from Jones' book on "Equilibrium and Vertigo," page 189:

> Summarizing, therefore, the cerebellum plays only a partial rôle in the large mechanism of the pointing. The kinetic-static sense, the arthroidal sense, tactile, auditory and visual impressions and memory all combine to inform the individual of the position of an external object. The motor areas of the cerebral cortex then send impulses to the arms; the function of the cerebellum is merely in controlling the execution of the cerebral mandate. If this be erroneous, because of the vertigo, past-pointing results.

The symptoms of dyssynergia, dysmetria and dysdiadokokinesia were marked and characteristic. They would tend to confirm our prevalent notions regarding cerebellar symptomatology. In contrast to the above symptoms was the conservation of trunkal static equilibrium as evidenced by the ease with which this patient maintained the sitting position. Mills and Weisenburg [8] insist that when trunkal movements are affected the vermis must be involved in whole or in part. The intact vermis and its related roof nuclei in the present case are again emphasized in this connection. Cerebellar catalepsy in this case is another example of conserved static equilibrium contrasted with disturbed dynamic equilibrium exemplified by the great incoordination in the extremities.

From the anatomic standpoint, the principal findings are corroborative in general of the degenerations following cerebellar defects such as are demonstrated in cases of cerebellar lesions, animal experimentation, and particularly unilateral and bilateral agenesias of the cerebellar hemispheres and reported by Edinger,[9] Anton and Zingerle,[10] and Oliver Strong.[11] But as emphasized by Anton, the degenerative changes following congenital defects are not necessarily identical with degenerations following later lesions because of the involvement of associated neurons in the former. Worthy of mention, and perhaps throwing some light on the anatomic connections of the cerebellum and of the inferior olive, are the following considerations:

1. The destruction of the left restiform body may be brought into relationship with the destruction of the left dentate nucleus for the reason that the right restiform body is practically intact with an extensive destruction of both cerebellar hemispheres and conservation of the right dentate nucleus.

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8. Mills, C. K., and Weisenburg, T. H.: Cerebellar Symptoms and Cerebellar Localization, J. A. M. A. **63**:1813 (Nov. 21) 1914.

9. Neuberger and Edinger: Berl. klin. Wchnschr. **35**:69, 1898.

10. Anton and Zingerle: Genaue Beschreibung eines Falles von beiderseitegem Kleinhirnmangel, Arch. f. Psychiat. **54**:8, 1914.

11. Strong, Oliver: A Case of Unilateral Cerebellar Agenesia, J. Comp. Anatomy **25**:361, 1915.

2. On the left side the degenerations in the central tegmental tract, circumolivary fibers, internal fibers of the olive, and Helweg's bundle seem to indicate a more or less intimate connection of these structures. Helweg's original anatomic studies [12] by means of carmin sections led him to the conclusion that his wedge-shaped spinal bundle was continuous above with the fibers surrounding the inferior olive and cephalad with his "oval" bundle, which is doubtless the tract now known as the central tegmental tract. Helweg believed that the fibers of the oval bundle formed the posterior commissure. The upward continuation of the degenerated central tegmental tract could not be traced in our case above the red nucleus. In the reported cases of agenesia above mentioned, there is no uniformity in the degeneration of these structures. In Edinger's case there was an absence of the right cerebellar hemisphere. The right central tegmental tract above the normal right olive was markedly defective. The degeneration was traced to below the anterior quadrigemina. Helweg's bundle was not mentioned. In the bilateral agenesia of Anton and Zingerle the central tegmental tract was not degenerated, but there was a bilateral degeneration of both Helweg's tracts. These authors draw a close relationship between these degenerations and the degenerated inferior olives. In Oliver Strong's case, in which the left cerebellar hemisphere was missing, the right central tegmental tract was degenerated, Helweg's bundle not being mentioned.

3. On account of the small tegmental lesion, which is in close vicinity to the central tegmental tract, the question must remain an open one as to whether this tegmento-olivo-spinal degeneration is dependent on it or on the cerebellar lesions. In a careful study of the serial sections representing the tegmental lesion no actual defect in the bundle can be demonstrated due to softening. This lesion probably explains the degeneration of the left median fillet which it directly involves. Regarding the significance of the above tegmental lesion and the other two small lesions in this case, we do not believe that they affect to any considerable degree the clinico-pathologic interpretations.

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12. Helweg: Stüdien über den centralen Verltuf der Vasomotorischen Nervenbahnen **19**:104, 1887.

A CASE OF ACROMEGALIA ASSOCIATED WITH BRAIN TUMOR

WILLIAM A. BRYAN, M.D., AND SHICHI UYEMATSU, M.D.
HATHORNE, MASS.

INTRODUCTION

The principal interest in this case is the relation between the cerebral tumor and the acromegalic symptoms. To prove which of these was primary or causative is impossible, but we will endeavor to show that the entire picture can be explained on the assumption that the tumor was the original lesion and the polyglandular symptoms were dependent on pressure with resultant biochemical changes. There is abundant support in the literature for this contention.

This patient has been under observation at various hospitals since March, 1914, with the diagnosis of acromegalia, the tumor mass not being suspected.

CLINICAL OBSERVATIONS

History.—J. B., Case 18457; Necropsy 2072. The patient was admitted to the Danvers State Hospital, Feb. 15, 1915. The family history was negative for nervous and mental diseases. The patient was born in 1865. Birth and early development were uneventful. He was married in 1890 and had six children. His wife had had no miscarriages. At the time his illness began, he was a foreman in a shop. He had no venereal disease. He used alcohol and tobacco moderately. His trouble began about 1895. The first symptoms were a sense of pressure and discomfort in the head with enlargement of the head, hands and feet, bowing of both legs and loss in weight. Progress was gradual, and the patient continued to work as a machinist. In the spring of 1903 he sustained a so-called shock. This caused paralysis of the left arm and leg and incapacitated him for about three months. In 1910 he began to have severe, almost constant headaches. At times they were diffuse but usually they were occipital. The pain in the hands became more acute. He remained at work until 1913 when he was finally compelled to resign. He became irritable and slept poorly.

He was admitted to the Psychopathic Department of the Boston State Hospital, March 19, 1914. Here he was cooperative and showed considerable insight, good orientation, very fair memory and no delusions or hallucinations. He was depressed and contemplated suicide because of his apparently hopeless condition.

Examination.—There was thickening of the sucutaneous tissues over the supra-orbital ridges, fingers, hands, toes, feet and nose. The hands were markedly enlarged and the tongue thick. The heart showed a systolic murmur. Exophthalmos was present, pupils and fundi were normal. There was a positive von Graefe sign. The left knee jerk was sluggish, the right was obtained only with reinforcement. Other reflexes were sluggish but equal. The patient had a waddling gait. Cutaneous sensibility and special senses

were unimpaired. The blood pressure was systolic, 140; pulse, 105; respiration, 20. The left lobe of the thyroid gland was enlarged. The Wassermann mann reaction with blood was negative; the spinal fluid was normal. Polydipsia and polyuria with vomiting had been persistent for six months before admission. The sugar content was 8 per cent.

Treatment and Course.—On April 18, 1914, a transsphenoidal operation was attempted. A large sella turcica was exposed and the bulging dura incised. Every attempt to excise the exposed gland led to such an amount of hemorrhage that no tissue was removed. On May 2 the wound was reopened and a radium tube inserted into the gland substance. The tube was left in situ for six hours. There was complete subsidence of his headache and the patient was discharged May 16.

Visual acuity was 20/15 in either eye, and there was only 0.3 per cent. sugar. The neuralgia on the right side had entirely disappeared, and the patient was free from headache and vomiting.

He returned to work until Thanksgiving, 1914, when he quit, principally on account of mental symptoms. He became more irritable, lost interest in his work, showed considerable memory loss, was drowsy, developed ideas of poisoning and thought his wife and daughter were persecuting him. He became violent at times and used obscene language. He threatened to commit suicide.

On admittance he was quiet in manner and cooperative. At times he became excited, spoke loudly and forcibly. There was no speech disturbance. For a few hours he was not clearly oriented. School knowledge and calculations were fairly accurate. His handwriting was normal. Memory was excellent. The patient talked clearly but at times showed some confusion. Apparently he had delusions of persecution; he complained of poison being placed in his food. He had no insight. He showed considerable irritability and at times was somewhat impulsive and hard to manage.

He was a moderately well nourished man. His nose was much enlarged and twisted to the right. The inferior maxilla was prominent. His hands were much enlarged, especially around the joints. His feet were enormous in size, the legs much bowed. He had hypertrophy of the tibia and femur, enlargement of the knee, wrist and metatarsophalangeal joints. There was increased fremitus at the right apex and a systolic murmur was transmitted to the axilla. There was marked sclerosis of the peripheral arteries. The systolic pressure was 155. The tongue was large and red. Urine: The specific gravity was 1.026; sugar, 0.4 per cent. The blood and spinal fluid Wassermann reactions were negative. There was a slight increase in globulin and albumin but the colloidal gold test was negative.

The left pupil was larger than the right; both pupils were moderately dilated and reacted fairly well to light and accommodation. Vision of the right eye was 20/40, of the left 20/50. The fundi showed a thickening of the retinal vessels and some evidence of choked disk in both eyes. Cutaneous sensibility and special senses were normal. Deep reflexes were absent; superficial reflexes were present and equal. There was a marked Romberg sign and considerable tremor of the hands.

Head Measurements: The head measurements were: biparietal, 15 cm.; occipitofrontal, 20 cm.; mentobregmatic, 27 cm.; occipitomental, 22 cm.; bitemporal, 11 cm.; intermastoid, 14 cm.; interzygomatic, 14.5 cm., and the cephalic index 75 cm.

The patient was up and dressed shortly after admittance. He was sent to the arts and crafts department where he became much interested in copper

work. He was pleasant, agreeable and cooperative. He complained of pain in the legs, a vague feeling of discomfort in his abdomen, and occasional headaches, diffuse in character.

He continued in this state until August, 1916, when he began to complain of increased dizziness which caused him to remain in bed, at first only a portion of the time, but later continuously. He became more unsteady in his gait and was unable to get about alone. Pain in the head increased, being more marked over the vertex. There was considerable loss of weight and strength. The left side was much weaker than the right. The deep reflexes on the right were greater. Hypertrophy of the face and joints and

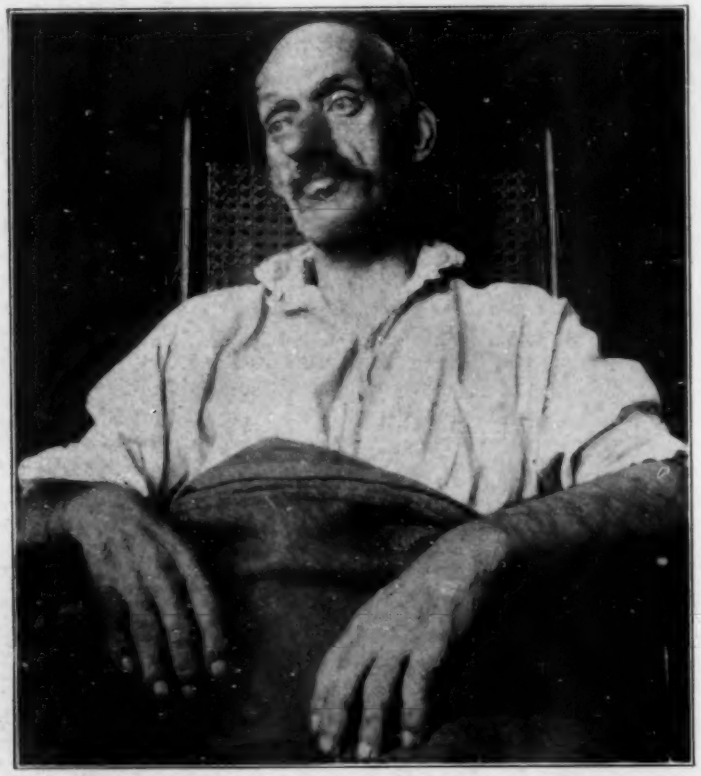

Fig. 1.—Patient seven months before death, showing characteristic changes of acromegaly.

bowing of the legs had increased. He was clumsy and awkward and there was considerable impediment in speech. Sensation was normal but hearing was better on the left. Optic atrophy was more pronounced on the right. He showed a rather childish pride in his condition with occasional attacks of irritability, but apparently the ideas of persecution had disappeared.

He continued to fail mentally and physically until he died, April 26, 1918, apparently of bronchopneumonia.

Postmortem Observations: Gross Anatomy.—Postmortem rigidity was present to some extent. The pupils were equal and regular, 4.5 mm. in

diameter. The ciliary vessels at the junction between iris and cornea were greatly injected on the right. The left eye was more prominent than the right. The nose measured 8.5 cm. in length and at the lower portion was 4.2 cm. in width. The supra-orbital crest was massive. The circumference of the head was 58 cm.; the distance from the top of the head to the mandible was 87.5 cm. The chin was prominent and greatly thickened. The transverse diameter of the head was 14 cm., the antero-posterior diameter 20 cm. The zygomatic processes and superior maxillary bones were greatly enlarged, the lips unusually thick. The forearms appeared slightly curved. The right wrist was 18.5 cm. in circumference, the left 18.7 cm. The terminal phalanges were greatly flattened. The measurements of the hands were: right hand: circumference, 23.8 cm.; index finger, length, 11.5 cm.; circumference middle phalanx, 8 cm.; circumference terminal phalanx, 7 cm.; middle finger, length, 11.5 cm.; circumference middle phalanx, 8.1 cm.; circumference terminal phalanx, 6.8 cm.; left hand: circumference, 22.5 cm.; index finger, length, 10.7 cm.; circumference middle phalanx, 7.5 cm.; and circumference terminal phalanx, 6.5 cm.

With heels together, the distance between the knees was 16.5 cm. There was marked bowing of the tibiae and the tibial crests were slightly roughened and flat, especially the left. The right ankle was 23 cm. in circumference, the left 23.5 cm. The right foot was 24.5 cm. long, the circumference at the ball of the foot 23.5 cm. The left foot was 24 cm. long, the circumference at the ball 24.5 cm. The toes were greatly enlarged and flat. There was exostosis at the joints. There was a heavy, hairy growth over the arms, pubis and legs; no adenopathy. The skin over the chest was very thick; there was practically no fat. Over the abdomen the fat measured 1 cm.

Head: Measurements: frontal, 1 cm.; temporal, 0.3 cm.; occipital, 0.8 cm. Above the interior occipital protuberance and to the right, there was an irregular, worm eaten erosion into the diploic space. The scalp seemed thinner than the external measurements indicated. The dura was adherent. The frontal sinuses were tremendously enlarged. The dura over the right occipital and parietal lobes had been destroyed and an irregular nodular hemorrhagic area was seen over the hemisphere along this side. The pituitary was rather firmly adherent to the sella turcica. The sella measured 2.5 cm. by 2.5 cm., and was 1.5 cm. deep. On either side of the sella floor was a depression into which the fifth finger fitted snugly. In the thin posterior wall of the sphenoid sinus was a round hole 0.6 cm. in diameter, which communicated with the nasal cavity.

Brain: Over the right parietal and occipital region there was a large, nodular, irregular tumor mass which was easily removed from the brain. It measured 8.5 by 9.5 cm. and consisted of about fifteen nodules, the smallest measuring about 1 cm. in diameter and the largest 7 cm. Some of them were darkly pigmented, others were white and glistening. The upper surfaces were firmly attached to the dura while the lower surface was smooth and covered by a thin layer of fibrous membrane. Capillaries in the pia were greatly injected. There was no sclerosis of the basal vessels. The olfactory bulbs were large but not adherent.

The whole parietal region was pressed down by the tumor mass which was separated by the pia mater from the brain substance. The tumor was not, therefore, derived from the brain substance but from the meninges. The markings of the convolutions in this depressed area had entirely disappeared and were not visible. The depressed convolutions were the upper part of

the anterior and posterior central convolutions, the paracentral lobule, superior parietal lobe and precuneus. The lower part of the precuneus, the posterior part of the cingular gyrus, the anterior part of the lingular gyrus, and the posterior part of the hippocampal gyrus were pressed out behind the splenium of the corpus callosum. The posterior two thirds of the cingular gyrus were pressed out over the corpus callosum, compressing the corresponding part of the left hemisphere. The third ventricle was thus pressed between the tip of the temporal lobe (which was also markedly compressed and flattened) and the posterior two thirds of the cingular gyrus. The occipital lobe was com-

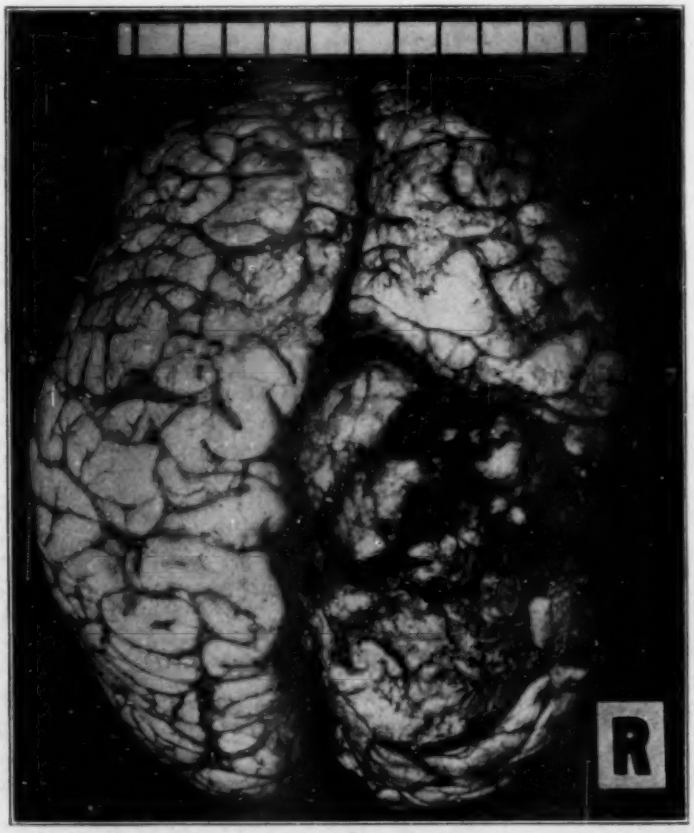

Fig. 2.—Superior surface of the brain showing tumor masses and flattening of convolutions.

pressed from the origin of the calcarine fissure to the tip of the occipital lobe, measuring 2.9 cm., while the left side measured 3.8 cm. The left hemisphere showed general flattening of the convolutions. The middle part of the median aspect of the left hemisphere showed marked concavity.

The corpus callosum was pressed down and the middle part appeared somewhat thinner. In consequence of pressure on the corpus callosum, the septum lucidum was folded up into three parallel wrinkles. The interventricular foramen was open. The body of the fornix was flattened. The intermediate

mass of the thalamus appeared flattened. The optic recess was somewhat distended, especially on the right side, and the optic nerves were markedly flattened on both sides. The infundibulum was pressed down. The pineal body and pineal recess seemed slightly compressed. The third ventricle was thus generally much flattened. Pressure of the tumor on the third ventricle seemed especially strong in the middle part, i. e., on the pituitary body.

From the details of the complete necropsy examination may be noted: Other findings corresponded to the clinical examination; they were negative

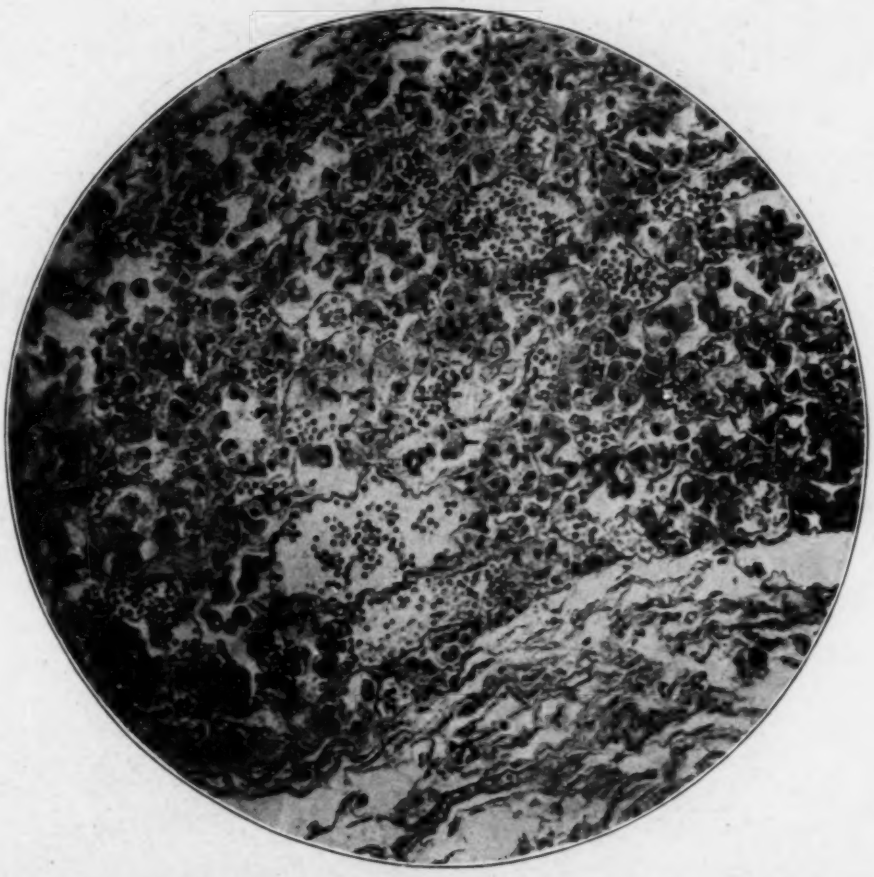

Fig. 3.—Histologic structure of the marginal zone of the pituitary body.

or unimportant. There were increase of pericardial fluid and chronic endocarditis; the coronaries were slightly sclerotic. The aorta was thickened and sclerotic. The liver showed a tendency to nutmeg appearance and fatty streaks. Chronic diffuse nephritis was present. There were yellowish spots in the cortex of left suprarenal, which was smaller than the right; the medulla of the right was thicker and there was a hemorrhagic spot beneath the capsule. The left lobe of the thyroid gland was nodular and hard and weighed 45 gm.; the right weighed 15 gm. The pons, midbrain and cord were soft.

Histologic Examination.—Heart: Fibers somewhat atrophic. Some fatty infiltration seen beneath epicardium. Aorta: Slight sclerosis. Lungs: Early bronchopneumonia; marked congestion. Spleen: Acute splenic tumor, hyperemic. Liver: Chronic passive congestion; central atrophy and necrosis; periportal increase of connective tissue, with lymphocytic infiltration. Pancreas: Marked chronic pancreatitis; sclerosis of many Isles of Langhan; fatty infiltration of some lobules seen. Kidneys: Cloudy swelling and marked congestion. Testicles: Paucity of interstitial tissue with few cells of Leidig;

Fig. 4.—Chromophobe struma, showing both marginal (relatively normal) and central zone.

parenchyma slightly atrophic. Prostate: Lymphocytic infiltration and some glands showed definite tendency to adenomatous transformation. Thyroid: Left lobe, marked increase of interstitial tissue and hyperplasia of epithelial cells with practically no colloid. Right, marked hyperplasia of parenchymatous elements with excess of colloid; tendency to interstitial proliferation. Suprarenals: Fatty degeneration and lymphocytic infiltration in zona fasciculata of the cortex. In the cortex were areas of adenomatous transformation. The medullar cells were pigmented.

Pituitary Body: The dural capsule was remarkably thickened. Most of the posterior lobe showed degeneration and was torn away when taken out. The anterior lobe was hyperplastic in its entire body except in a narrow marginal zone. This marginal zone presented still sinusoidal structure with luxuriant vascularity. Almost all cells were neutrophilic. The rest of the pituitary consisted of a more or less loosely packed body of epithelial elements with no demonstrable connective tissue and practically no blood vessels. Toward the center of the lobe the cell elements were arranged more

Fig. 5.—Histologic picture of the endothelioma.

irregularly and were widely separated. Except in the marginal zone no sinusoidal or alveolar structure was discerned. In the center of the gland practically all cell elements were neutrophilic. In a part of the hypertrophic central region there was evidence of increased secretion of colloidal matter, the perivascular spaces being filled with a pinkish stained homogeneous matter. No mitotic figures were observed.

The condition of the pituitary body was no doubt pathologic, exhibiting adenomatous characteristics. It corresponded with the chromophobe struma of Cushing's nomenclature and also with the malignant adenoma of early writers.

Histology of the Tumor.—The tumor was made up of long endothelial cells with elongated nuclei. They were rather regularly arranged showing a parallel course and a concentric arrangement around the vessels. The vessels were rather scant. Although the cells were mostly elongated, there were a number of different forms. Some were round, the nuclei being fat and regular in shape. They were not arranged regularly, but showed a bizarre order. The nuclei of the tumor cells showed also a remarkable polymorphous condition. Some

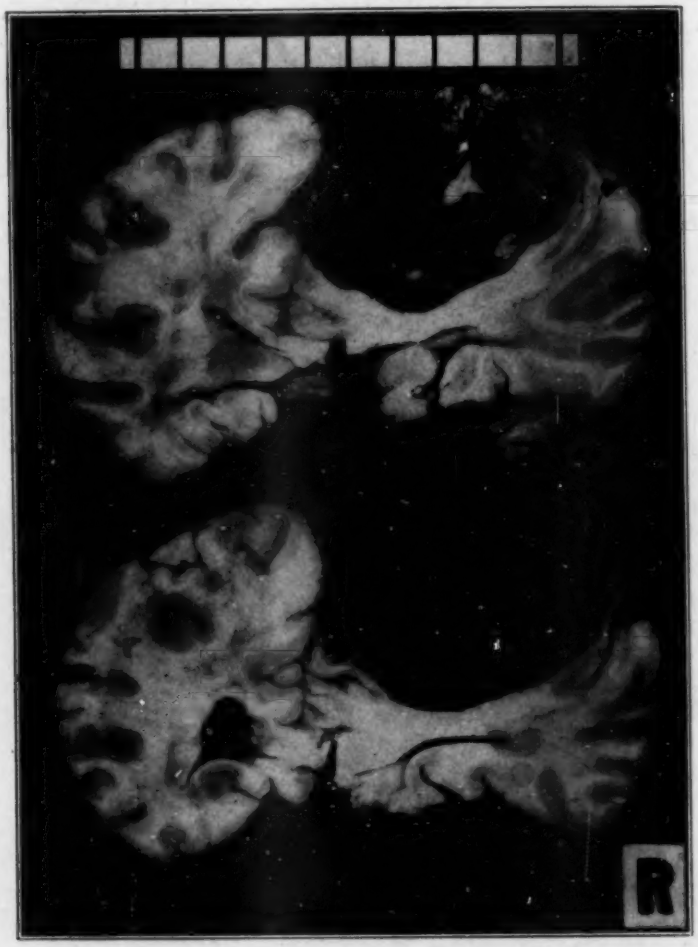

Fig. 6.—Cut surface of the brain according to Dalton's method.

were round and small, others oval, spindle, rod shaped and much elongated. A great number of nuclei showed direct division, while many showed shrinkage, vacuole formation, etc. Apparently there had been both a proliferating and a regressive process going on simultaneously, with the former progressing more slowly.

Observation of Cut Surface of the Brain According to Dalton's Method.—The herniation of the right gyrus fornicatus was well observed in the cut sur-

faces. The right temporal lobe was much compressed and flattened. The right lateral ventricle was practically obliterated. The left ventricle was also remarkably compressed in its middle part where it was seen only as a narrow crevice.

COMMENT

The symptoms of this patient may be divided into those of acromegaly, exophthalmic goiter, cerebral tumor and mental manifestations due to organic lesion.

Acromegalic Symptoms.—Marie represents the view which attributes acromegaly to diminished function of the pituitary body. Strümpell, Guerrini, Cagnett and others believe that this disease is due to nutritional disturbances, the enlargement of the pituitary being only secondary. The hypertrophy of the gland is regarded by others as even accidental. Massalongo, Tamburini, Benda, Modena, Fisher and Cushing favor the hyperpituitarism conception because of the usual finding of glandular hypertrophy, the opposite clinical manifestation from experimental extirpation of the anterior lobe, the low carbohydrate assimilation in the earlier stage of the disease, improvement of the disease from a partial extirpation of the hyperplastic gland, etc. A great many authors have observed hypertrophic enlargement of the gland and a histologically demonstrable hyperplasia, and it is probable that the latter view is correct.

In the present case, as in most others, a condition which was called by Cushing "chromophobe struma" was found; also evidences of hypersecretion.

The adenomatous transformation of the gland in this case, however, is not to be considered as "malignant" in the sense used by earlier observers. We regard it merely as an extreme hyperplastic condition of the gland, for these reasons:

1. In spite of the long standing disease the pituitary was only slightly enlarged and retained its original form.

2. The capsule was thickened and showed no tendency to rupture by the proliferated gland substance.

3. The marginal zone remained practically unaltered.

Cushing seems to hold the same opinion with regard to the nature of the hyperplasia. "It would appear," he says, "that any of these functionally unstable glands may, under certain biochemical stimuli, assume adenomatous characteristics, and doubtless most individuals afflicted with acromegaly face the possibility of such a transformation." We have in the present case, a definite histopathologic basis to account for the cynical syndrome, and we believe that this condition was responsible for the various symptoms.

Although it was impossble to examine the posterior lobe on account of the softening, it is obvious that this had nothing to do with the acromegalic syndrome. The syndrome of adiposity, high sugar tolerance, subnormal temperature, slow pulse, asthenia, etc., is accounted for by posterior lobe deficiency. The present case did not show any of these symptoms. The degeneration of the posterior lobe must, therefore, have been of late occurrence.

Exophthalmic Goiter.—In no one of Cushing's forty-seven cases was there symptomatic evidence of hyperthyroidism. Cushing admits that some degree of exophthalmos is common in hypophysial disorders, but he attributes this to the neighborhood effect of the glandular tumor. In our case the patient showed not only exophthalmos, but also struma, von Graefe's sign, tachycardia, etc., and they are, in all probability, to be regarded as evidence of hyperthyroidism. Moreover, the pituitary body was only slightly enlarged and no neighborhood symptoms were noted.

The thyroid was enlarged and asymmetrical. Microscopically, parenchymatous hypertrophy with luxurious vascularization, was demonstrated. There was also increase in the interstitial tissue of both lobes. The findings in the thyroid correspond to those of a typical case of exophthalmic goiter of long standing. The findings in the thyroid were entirely different from those of myxedema, although there was remarkable increase in the interstitial tissue. The latter shows an increase in old cases of exophthalmic goiter.

Cushing and others have reported cases with a polyglandular syndrome in which the symptoms of hyperadrenalism and hypo-adrenalism were apparent. It is quite possible, therefore, for the thyroid to show secondary symptomatic manifestations as a result of a lesion in the pituitary body.

Cerebral Tumor.—Microscopic examination showed the tumor mass to be typical endotheliomas. This tumor is of slow growth. Cushing describes two cases in which the lesion had been causing local symptoms for thirteen and nine years, respectively, before general pressure symptoms appeared. Most of the symptoms of brain tumor were observed in this case. About eight years after the onset of the disease, the patient had a so-called shock followed by paresis of the left arm and leg. This is to be regarded as a focal manifestation of the tumor. Four years before his death choked disk was discovered. During the last four years he suffered from constant headaches and periodical vomiting.

No doubt the diagnosis is clouded by the symptoms of acromegaly. However, if both general and focal manifestations had been thor-

oughly studied, it might have been possible to make a correct diagnosis, at least in the later stages of the disease.

Mental Symptoms.—Irritability, distrust, indecisiveness and lack of concentration are attributable to the hypophysial derangement. Lack of interest, drowsiness and deterioration are probably due to increased intracranial pressure. The depression in the early stage of the disease, on the other hand, might have been nothing but physiologic reaction to his hopeless condition.

The Cerebral Tumor and Pituitary Body Disorder.—In the literature are a great many cases of acromegaly with cerebral tumor situated in the neighborhood or at a distance. Are the tumors found in these cases accidental, having nothing to do with the disturbances of the pituitary body? Cushing seems to be inclined to believe in the etiologic significance of these cerebral tumors in the development of hyperpituitarism and hypopituitarism, stating: "In every case of increased intracranial tension, from whatever source, there probably occur secondary changes in the hypophysis, often with gross deformations and resultant functional disturbances which frequently elicit recognizable clinical manifestations." Cushing observed a case of outspoken acromegaly with an unsuspected cerebellar cyst. "Whether the hypophysial hyperplasia" he says, "was merely a concomitant process which bore no relation to the obstructive hydrocephalus, or whether the gland had been aroused into its state of pathologic overactivity as a secondary result of the cerebellar lesion cannot be positively certified. I incline toward the latter view."

In our case the third ventricle was not especially dilated, but the pressure of the tumor had been exercised directly on the hypophysial fossa. Apparently the endothelioma dated back before the symptoms of acromegaly appeared. Whether or not the tumor had been playing a causative rôle is difficult to determine. However, it seems to us that it is quite possible for the brain tumor to have caused secondary changes in the pituitary with consequent manifestations of acromegaly.

The Relation of Acromegalia and Exophthalmic Goiter.—In most cases of acromegalia in the literature the thyroid gland was found more or less altered. It showed either hypertrophy or atrophy. In experimental extirpation of the pituitary body the thyroid seems to show transient hypertrophy followed by colloid degeneration. This condition can not be explained by the vicarious function of similar organs of the body. Cushing observed in most cases of acromegalia enlargement of the thyroid instead of atrophy, and he suggests the "same underlying biochemical factor" causing hyperplasia of both structures at the same time.

If the biochemical factor of Cushing's hypothesis is accepted, the hyperplastic condition of endocrine organs in our case is readily explained. But what is the "biochemical factor"? If the existence of this so-called biochemical factor is primary and the changes in the pituitary and other endocrine organs are secondary, acromegalia is not an independent disease but a partial manifestation of some pathologic metabolism that gives rise to a biochemical stimulus. If this exploitation of the relationship of the endocrine organs in our present case were accepted the mechanical pressure of the cerebral tumor would no longer be responsible for the production of the acromegalia. We are rather of the view that the hypertrophy of thyroid and other endocrine organs is secondary, due to functional alteration of the pituitary body. Although the thyroid gland and the pituitary body show histologic and functional similarity, there is no definite evidence that these organs necessarily function in a synergic manner. Both may show hypertrophy without a common biochemical stimulus, but in consequence of the altered function of one of them. It is possible that pathologically increased function of the anterior lobe of the pituitary gland exercises a certain influence on the secretory organs of the body, causing changes in these organs in the same direction.

We maintain that the cerebral tumor was, in all probability, the cause of the acromegalia in this case. First, the pituitary body was attacked showing both the clinical and pathologic picture of typical acromegalia. Second, the endocrine organs were involved in consequence of the altered function of the hypophysial gland. The exophthalmic goiter is to be considered as a clinical manifestation of the pathologic thyroid gland.

Danvers State Hospital.

MOTOR DISTURBANCES IN LETHARGIC ENCEPHALITIS *

ISADOR ABRAHAMSON, M.D.
Associate Neurologist, Mount Sinai Hospital; Visiting Neurologist, Montefiore Home
NEW YORK

We have seen that the main stress of the toxemia of lethargic encephalitis falls on the nerve cells. At certain sites the toxic action is reinforced by the direct attack of the infectious agents (Loewy-Strauss bodies?) on the nerve cells. The nerve sites attacked by these organisms are determined in part by the particular mucous membrane in which the initial invasion takes place. From this area, the organisms are carried along the lymphatics to the central nervous system. As the habitually invaded area is the nasopharyngeal mucosa, the nerve sites usually attacked are in the head end of the nerve axis; but if the intestinal mucosa be the invaded area, the brunt of the attack falls on the caudal end of the nerve axis. The direction, extent and severity of the attack on the nervous system determine the nature and degree of the resulting functional disturbances. It has already been stated that the more extensive the nervous mechanism on which a function depends, the more inevitable is the implication of that function in the attack. The nervous mechanism that subserves the function of movement is coextensive with the nervous system. In lethargic encephalitis movement is invariably deranged.

Lethargic encephalitis may affect the function of movement in all predictable ways, to all conceivable degrees and at all possible points of the motor mechanism. In different epidemics, and even during the same epidemic in different localities, or at different periods, particular varieties of motor derangement tend to prevail. In spite of such episodal variations among the qualities of the motor defect of lethargic encephalitis are certain that are banal and certain that at least in their emphasis are distinctive and pathognomonic.

Of any two movements, that which depends on the more extensive nerve mechanism is—ceteris paribus—the more liable to derangement in this infection. Where separate paths convey habitually correlated impulses to an associated group of muscles, sudden interference with any one path may disorganize temporarily the whole function. The

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initial result of the lesion may, therefore, be wholly disproportionate to its cause; and this disproportion may be accentuated if, as in the eye, the impairment of movement deranges another and more sensitive function.

MOTOR SYMPTOMS

Motor Disturbance of Eye.—The widespread nerve supply of the motor mechanism of the eye seldom wholly escapes implication in lethargic encephalitis. Double vision, near vision, blurred vision and other perceptual consequences may signify such motor implication even before it is detectable by skilled medical observation. The alternative paths of nerve impulses to the muscles of the eye may permit the speedy compensation of this functional disturbance. Hence, patients frequently neglect to mention it among their symptoms, unless specifically questioned regarding it.

In addition to these transient symptoms, lasting signs appear of impairment of the motor mechanism of the eyes. As a rule, the eyelids droop, first one, then the other. The droop is not commonly equal on both sides, and seldom completely closes both eyes; usually the palpebral fissures persist, one narrower than the other. In most cases the eyelids can be freely raised at will; but they fall again. Sometimes, however, they are not raised; but the patient then seems rarely to exert himself to open his eyes—the forehead is not wrinkled by his efforts.

The eyes assume the "rest" position; their axes being directed straight forward. They can be rotated as a rule into any normal position; but they do not maintain it; they tend to swing back to the middle line. Internal convergence is usually possible for a short time, at the end of which one eye rotates out again. The eyes may move deliberately and by stages, moving and halting and moving again, then swinging back to the position from which they started. Sometimes this movement is definitely cog-wheel, like the eye movement seen in Parkinson's disease. Infrequently, definite but seldom conspicuous squints occur.

The pupillary reaction to light and accommodation may be lost or diminished. Usually both are unaffected. The pupils under a constant intensity of illumination contract and dilate, unable to maintain the pupillary posture corresponding to the illumination. A similar instability may be observable in accommodation.

Facial Muscles.—The facial muscles are often affected. Spasms, tics and fibrillation may sometimes be seen in them and occasionally nuclear paralysis. As a rule the facial affection consists chiefly of a loss of tone, a flattening of the muscles, with a fading of the wrinkles, and a lessening of the habitual facial folds. This tone

loss begins on one side and then spreads to the other. It is usually not complete or symmetrical; on one side it may affect the upper third, on the other the lower two thirds. It tends to disappear in emotional expression. It suggests a supranuclear palsy, but the apparent weakness may vanish in whole or in part on voluntary movement. Occasionally, even in patients who are not extremely apathetic, a partial or complete absence of emotional expression is observable in a face that otherwise shows no detectable motor defect. The facial weakness, no matter what its type may be, is seldom sufficient to lead to lacrimation, to drooling or to interference even with mastication.

Fifth Nerve.—Occasionally, the motor root of the fifth nerve suffers. During the irritative stage, chewing or grinding movements occur, and later lock-jaw may supervene. Even without premonitory movements, the jaw muscles may gradually stiffen till the mouth scarcely can be forced open. This irritative stage may be followed by a typical paralytic condition.

Tongue.—The tongue is implicated almost as frequently as the face. After a period of fibrillary twitching, the tongue on protrusion deviates to the weakened side; but this deviation bears no regular correspondence to any coexisting facial palsy, except in banal cases of hemiplegia. Later, the tongue is protruded with increasing difficulty or lies an inert or tremulous mass, immovable on the floor of the mouth. In such instances mastication and speech suffer.

The Soft Palate and the Constrictors of the Pharynx.—These may be so weakened as to render swallowing difficult or even impossible and to necessitate feeding by the stomach tube.

Cranial Motor Nerves.—Of the cranial motor nerves the eleventh (spinal accessory) is least commonly affected. When it is attacked, there may be wry neck but more frequently both sides are equally involved. Associated with the spasms, fibrillation, or loss of tone of the trapezeii and sternomastoids are similar affections of the other muscles of the neck region. Cephaloptosis or cephaloplegia, similar to that seen in poliomyelitis, is occasionally observed in lethargic encephalitis, especially in children.

As the cranial motor nerves are affected, in like manner the disease attacks the spinal motor nerves; and the consequences observed in the muscles of the head and neck are mutatis mutandi, observed in the muscles of the limbs and trunk. In the affected muscles spasms, fibrillations, weakness and loss of tone usually occur. The weakness may follow the spasms but oftener develops independently. Foot drop, wrist drop, sagging shoulder and other abnormal attitudes are assumed in the "resting" position. The dropped foot, wrist or shoulder, as a rule, can be raised at will; but it quickly drops again.

Hemiplegias and Monoplegias.—Not rarely banal hemiplegias or monoplegias occur. In these the distal muscles of the extremities are more involved than the proximal. Some power of movement usually persists in the trunk muscles of the affected side and in the muscles nearest the trunk in the affected limbs. The affected muscles are rigid and immobile. They assume the characteristic hemiplegic attitude, which is determined mainly by the dominance of the flexors over the extensors. Occasionally athetoid movements are seen in the paralyzed limbs.

Besides this banal hemiplegia, rigidity and loss of power of lesser degree occur, often hemiplegic in distribution. The rigid muscles, partially immobilized, tend to assume the typical hemiplegic posture and tremors appear in them. The characteristic tremor in lethargic encephalitis, like the tremor of Parkinson's disease, visibly affects extremities more than the head and trunk and is exaggerated by emotional excitement and by indirect intention. The lethargic encephalitis tremor, however, is not present "at rest"; it is markedly increased by direct intention and grows in amplitude as the objective of the movement is approached; it involves the forearm and wrist more than the hand and finger joints and the leg and ankle more than the toes; it is coarser, less symmetrical and more irregular than the typical Parkinsonian tremor. Where such a tremor ends, and a rhythmic movement purposeful in form but not in intent, begins, is difficult to define. The coarse pseudoparkinsonian tremor of lethargic encephalitis is scarcely distinguishable from a slight, irregular rhythmic purposeful movement.

In addition to this coarse tremor, the small, rapid tremor seen in most acute infectious diseases, occurs also in lethargic encephalitis.

Occasionally epilepsy develops.

The most characteristic of the motor disturbances is instability of posture.

LOSS OF MUSCLE TONE AND ITS RELATION TO POSTURE

Posture is the expression not of one, but of all the influences that determine muscle tone. The fundamental influence is the nutrition of the individual muscle cell, which is dependent partly on general nutritive conditions and partly on local and general control of these conditions through reflex and chemical action. In lethargic encephalitis loss of muscle tone occurs.

Part of this loss is analogous to that which occurs in all acute infectious diseases. Fever, toxemia, and their concomitant endocrine disturbances always reduce muscle tone. With the development of acute infectious fevers reflexes, such as the knee jerks, which depend on muscle tone, diminish or disappear; with the abatement of the

infection the muscle tone returns and the reflexes related to it recover their activity. Among infectious fevers lethargic encephalitis is conspicuous by its power directly to lower muscle tone, perhaps because of the virulence of its toxins, perhaps because of their inhibitory action on the pituitary and suprarenal glands.

Part of the loss may be due to inflammatory implication of nerve centers and paths by which the tone of the musculature is regulated. In lethargic encephalitis lesions may occur in the vestibular and cerebellar systems through which local, sectional and general changes in muscle tone are initiated and correlated. But the tone loss is common and such lesions are infrequent.

Part of this loss may be due also to the injury or destruction of the lower motor neuron, either at its origin in the nuclei of the anterior horn of the spinal cord and in their pontile and medullary analogues, or during its course through the peripheral motor nerve. A preliminary stage of irritation associated with muscular rigidity and fibrillation and myoclonia sometimes may be observed; then atonicity, atrophy and quantitative or qualitative changes in the electrical reactions occur. This loss is seldom widespread. It does occur, but it is often absent. Habitually, the electrical responses to faradism and to galvanism are unaltered. These responses are given by the fibrillar portion of voluntary muscle; which is therefore not blamable for the customary loss of muscle tone in lethargic encephalitis.

The tone loss is evident in the afflicted muscles even in so-called "rest" postures. There is in life no such thing as a posture of absolute "rest." "Rest" is a purely relative term. It signifies usually a posture of mental repose, the posture that is determined chiefly by the action of gravity. Its maintenance requires definite degrees of muscle tone. Hence, the afflicted facial muscles "at rest" flatten, the wrinkles fade and the usual facial folds disappear. In other words, owing to depreciation of muscle tone, the normal facial posture "at rest" is lost.

When a muscle contracts its tone increases. The increase of tone that accompanies voluntary movement may be adequate to mask the tone loss of the "resting" muscle. Thus, the atonic facial muscle sometimes moves at will with seeming perfection; but sometimes the flattening persists. The amount of disturbance persisting in movement measures the failure of the increased tonicity of the voluntary moving muscles to mask the tone loss of the "resting" muscle.

In moving, a muscle proceeds from an existing attitude through a series of intermediate postures to the desired attitude. To every posture pertains a definite muscular tone. Throughout a movement the tone of each posture must be maintained until that of its successor in the movement can be superimposed. Otherwise the desired

attitude is not directly attained and the movement is oscillatory or jerky. Lack of tone inherent to the intermediate postures leads to the phenomena seen when the eyes move—the slow rotations by stages and the persistent tendency to swing back to the "rest" position. It is as if the increased tone excited by the voluntary movement sufficed to attain the serial posture but not to preserve it. Hence, the eye swings to and fro in its rotation toward the desired goal.

And when the desired posture is attained by the eyes, lack of the necessary tone cuts short its duration. The eyelids can be raised at will but they fall again; and as often as they fall they can be raised once more only to fall once more. On internal convergence, an eye soon swings away. It may be brought in again only to swing out once more. With a constant source of light the pupil dilates and contracts. The outstretched arms may fall and be raised at will only to fall again.

This inability to maintain posture is not a fatigue manifestation, although fatigue may magnify it. It is not comparable with the similar phenomenon seen in myasthenia gravis. It is not a paralytic phenomenon. The movements can be indefinitely repeated and the same phenomenon is seen at the beginning as at the end of the test. The muscles show neither myasthenic nor atrophic electrical reactions. The lack of power to preserve posture is not due to the general lowering of tone for it affects only individual muscles or groups in particular areas.

In any movement certain muscles, agonists, contract, and they may be aided by the simultaneous contraction of others (synergists) which act similarly. Synchronously, the antagonistic muscles actively relax. If the antagonistic muscles do not adequately relax, the contracting agonists must first overcome the pull of the unrelaxed antagonists before movement can begin; and the movement proceeds by jerks and oscillations according to the momentary dominances of the agonists or antagonists in their contest. Such a struggle for dominance may occur. The evidence of it is spasms of the antagonistic muscles. Even without appreciable spasm there, the inability to maintain posture may exist. The force of gravity may suffice to destroy the posture attained.

When a posture is achieved it is maintained against gravity mainly by virtue of the tone inherent in the sarcoplasmatic portion of voluntary muscle. The sarcoplasmatic portion of voluntary muscle is distinguished from the fibrillar by its histologic structure, chemical composition, electrical reaction, function and nerve supply. The nerve supply is derived from neurons which originate in cells in the lateral horns of the spinal cord and their analogues in the cerebral end of the nerve axis. Through these nerves the sarcoplasmatic tone is

maintained on which depends the posture of the muscles. In lethargic encephalitis the nerve mechanism which subserves the sarcoplasmatic portion may be implicated, giving rise to their particular form of loss of power to maintain posture. Depression of the tone of the sarcoplasmatic portion of the agonists is probably the basis of this postural fault; but the tone of the antagonistic muscles may also be unduly exalted, for although spasm may be absent, the movements may nevertheless be somewhat stiff. At present, we do not know with certainty the precise seat of the defect. Investigation of it may afford valuable data regarding the nature of the relaxation of the antagonist and its relation to sarcoplasmatic tone.

It is noteworthy that the patient sometimes makes no effort to restore the lost posture. As there is no detectable sensory loss the lack of effort is probably due to mental apathy. This postural defect may arise when the lower motor neuron is demonstrably intact. It affects postures voluntarily assumed. There must exist, therefore, not one final common path to voluntary muscle, as Sherrington teaches, but two paths, namely, the lower motor neuron, its cell in the anterior horn and its white medullated fiber passing to the voluntary muscle, and the postural nerve of voluntary muscle. In poliomyelitis the lower motor neuron is mainly and commonly affected, and the postural nerve as a rule escapes. In lethargic encephalitis the lower motor neuron as a rule escapes and the postural nerve is mainly and commonly affected.

THE OCULOCARDIAC REFLEX (DAGNINI-ASCHNER PHENOMENON)—ITS USE IN MEDICINE AND PSYCHOLOGY

AN EXPERIMENTAL AND COMPARATIVE STUDY OF GROUPS OF NORMAL AND PATHOLOGIC SUBJECTS

SANTE NACCARATI, M.D., Sc.D.
NEW YORK

This phenomenon was first reported by Dagnini [1] at the meeting of the Academy of Medical Sciences of Bologna, June 17, 1908. Four months later B. Aschner,[2] unaware of the work of Dagnini, published a paper on the same subject. The reflex is called Aschner's reflex ordinarily, but in a spirit of fairness and justice to the Italian scientist, it should be identified primarily with the name of Dagnini.

The phenomenon consists in slowing of the radial pulse, lowering of the blood pressure and modification of the respiratory rhythm from compression of the eyeballs. Long before Dagnini and Aschner, Luciani had observed that stimulation of the branches of the trigeminal nerve with choloroform produced retardation of the heart; and Wagner von Jauregg used compression of the eyes to arouse stuporous patients. He did not explain the cause or the mechanism of the phenomenon.

Experiments on animals, carried out by Aschner, Miloslavich [3] and others, showed that this was a real reflex having as the centripetal pathway the trigeminal nerve and as the centrifugal pathway the vagus. Aschner also demonstrated that the phenomenon could not be attributed to stimulation of the vagus by increased intracranial pressure.

Petzetakis [4] and more recently Fumarola and Mingazzini [5] have shown that the centrifugal pathway of the oculocardiac reflex is constituted also, although in a lesser degree, by the sympathetic. By

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1. Dagnini, G.: Intorno ad un riflesso provocato in alcuni emiplegici collo stimolo della cornea e colla pressione sul bulbo oculare, Boll. di scienze med., Bologna **8**:380, 1908.

2. Aschner, B.: Ueber einen bisher noch nicht beschriebenen Reflex vom Auge auf Kreislauf und Atmung. Verschwinden des Radialispulces bei Druck auf das Auge (Vorläufige Mitteilung), Wien. klin. Wchnschr., No. 44: 1529 (Oct. 29) 1908.

3. Miloslavich, E.: Ueber Trigeminus- Vagusreflexe, Wien. med. Wchnschr. **51**:3051 (Dec. 17) 1910. Grossmann, J., and Miloslavich, E.: Ueber die Beeinflussung der Hertztätigkeit durch Bulbusdruck, Wien. klin. Rundschau **12**: 177, 1912.

4. Petzetakis: Etude expérimentale sur les voies centrifuges du réflexe oculo-cardiaque, Compt. rend. Soc. de biol. **76**:657 (April 25) 1914. Effets de la section de la moelle cervicale sur le rythme cardiaque, Arch. d. mal. du coeur **10**:66 (Feb.) 1917.

5. Fumarola, G., and Mingazzini, E.: Contributo clinico e sperimentale allo studio del riflesso oculo-cardiaco, Policlinico, Rome **24**:404 (Oct.) 1917.

severing the cervical cord, the sympathetic connection with the medulla is interrupted and the centrifugal impulse traveling only through the vagal cardio-inhibitory fibers causes exaggeration of the reflex.

Since the appearance of Daginini's and Ascher's works a great number of papers have been published on this subject. Many reports have been contradictory and misleading and the inversion or exaggeration of the oculocardiac reflex was given too broad a significance.

The reflex has also been employed therapeutically in paroxysmal tachycardia (Lian,[6] Voisin and Benhamou [7]) and in hiccough (Loeper and Weil [8]).

It has been assumed by some authors that the normal reflex is retardation of the pulse five to twelve beats per minute. When the pulse is reduced more than twelve the reflex is called exaggerated. When the retardation does not surpass four, the reflex is abolished. When instead of retardation, acceleration occurs, the reflex is inverted. Others have proposed similar classifications but, as will be shown later, classifications of this sort are arbitrary and do not give the real individual value because the same subject may show at different times a normal, an inverted and an abolished reflex, or a normal, an abolished and an exaggerated reflex. Small wonder that the reports of different authors are so discordant. Only in regard to tabes do the reports agree, the reflex having quite generally been found abolished by Auer,[9] Fumarola and Mingazzini,[5] Gautrelet,[10] Lesiur, Vernet and Petzetakis,[11] Levine,[12] Leeper and Mougeot,[13] Orlandi,[14] Santiago Barabine [15] and by the writer.

6. Lian, C.: De l'emploi thérapeutique du réflexe oculo-cardiaque dans les crises tachycardiques, Arch. d. mal. du coeur. **8**:193 (July) 1915.

7. Voisin, R., and Benhamou: La valeur thérapeutique du réflexe oculo-cardiaque. Paris méd. **9**:210 (March 8) 1919.

8. Loeper and Weil: Action favorable de la compression oculaire sur certaines manifestations nerveuses et en particulier sur le hoquet, Bull. et mém. Soc. méd. d. hôp. de Paris **37**:631, 1914.

9. Auer, E. M.: The Oculocardiac Reflex in Syphilis of the Central Nervous System, J. A. M. A. **68**:901 March 24) 1917.

10. Gautrelet, J.: Le réflexe oculo-cardiaque, Paris méd. **3**:583, 1912.

11. Lesieur, Vernet and Petzetakis: Note sur l'abolition fréquente du réflexe oculo-cardiaque dans le tabes, Bull. et mém. Soc. méd. d. hôp. de Lyon **13**:198 (March) 1914.

12. Levine, S. A.: The Oculocardiac Reflex. An Electrocardiographic Study with Special Reference to the Differences Between Right and Left Vagal and Ocular Pressure in Tabetics and Non-Tabetics, Arch. Int. Med. **15**:758 (May 15) 1915.

13. Loeper, M., and Mougeot, A.: Absence fréquente du réflexe oculo-cardiaque dans le tabes, Bull. et mém. Soc. méd. d. hôp. de Par., Dec. 26, 1913, p. 942; Progrès méd., Dec. 27, 1913, p. 675.

14. Orlandi, N.: Sul valore clinco del riflesso oculo-cardiaco, Riforma med. **31**:232, 260 and 288, 1915.

15. Santiago-Barabino, A.: Contribucion al estudio del reflejo óculo-cardiáco, Prensa med., Argentina, Buenos Aires, Nos. 29, 30, 31, 1917.

To give a few examples of the conflicting results in general paresis, Lesieur, Vernet and Petzetakis usually found the oculocardiac reflex exaggerated, while Fumarola and Mingazzini, Roubinovitch and Régauld de la Sourdière,[16] and Aguglia [17] found it abolished. In epilepsy Lesieur, Vernet and Petzetakis,[18] Aguglia, and Dufour and Legras [19] reported exaggeration of the reflex, while Maillard and Cordet [20] did not find it exaggerated and rejected the explanation of Lesieur, Vernet and Petzetakis that bromids cause the reflex to be less accentuated. In my experiments, although there was found a definite relative tendency to vagotonic reaction among the group of fifty epileptic patients, the findings of Lesieur, Vernet and Petzetakis could not be substantiated. Fumarola and Mingazzini [5] found in epileptics a tendency toward exaggeration. Orzechowski and Meisels [21] studied a group of epileptic patients with pharmacodynamic tests and found that they gave a vagotonic reaction. In diphtheria Aviragnet, Dorlencourt and Bouttier [22] found that of twenty-six patients the reflex was normal in 42.3 per cent. and abolished in 57.7 per cent., while Gunson,[23] who examined fifty cases, found that the reflex was normal in 92 per cent. of the patients. In dementia praecox, Truelle and Bourdelique [24] found the reflex inverted or almost so. Roubinovitch

16. Roubinovitch and Sourdière, Regnauld de la: Le réflexe oculo-cardiaque dans les demences organiques, Soc. psych. de Par., June 18, 1914.

17. Aguglia, E.: Il riflesso oculo-cardiaco negli alienati di mente, Riv. ital. di neuropatol., psichiat. ed elettrater **7**:385, 1914. Il riflesso oculo-respiratorio negli alienati di mente, Ibid. **8**:57, 1915.

18. Lesieur, Vernet and Petzetakis: Contribution à l'etude du réflexe oculo-cardiaque; son exagération dans l'epilepsie; ses variations sous l'influence d'actions médicamenteuses ou toxiques, Bull. et mém. Soc. méd. d. hôp. de Par. March 6, 1914; Considérations sur les modifications des reflexes produits par compression des globes oculaires chez certains epileptiques, Lyon méd. March 29, 1914, p. 721; Considérations physio-pathologiques sur un cas d'arrêt du coeur par le réflexe oculo-cardiaque chez un épileptique, Bull. et mém. Soc. med. d. hôp. de Par. **37**:394 (March 6) 1914.

19. Dufour, H., and Legras, M.: Réflexe oculo-cardiaque provoquant l'arrêt du coeur, l'automatisme ventriculaire et la dissociation auriculoventriculaire; syndrome hypovarien et hyperthyroiden; crises épileptiformes; Bull. et mém. Soc. méd. d. hôp. de Par. **37**:686 (April) 1914.

20. Maillard and Codet: Le réflexe oculo-cardiaque chez les epileptiques, Soc. de Psychiat. de Par., June 18, 1914.

21. Orzechowski, K., and Meisels, E.: Untersuchung über das Verhalten des vegetativen Nervensystems in der Epilepsie, Epilepsia, Leipzig **4**:181 and 293, 1912-1913-1914.

22. Aviragnet, E. C.; Dorlencourt, H., and Bouttier, H.: Le réflexe oculo-cardiaque au cours de l'intoxication diphtérique, Compt. rend. Soc. de Biol., **76**:771 (May) 1914.

23. Gunson, E. B.: The Oculo-Cardiac Reflex, Brit. J. Child. Dis. **12**:97, 1915.

24. Truelle and Bouderlique: Le réflexe oculo-cardiaque dans la dèmence précoce des jeunes gens, Ann. méd. psychol. **7**:389, 1916.

and Regnauld de la Sourdière found the reflex abolished in 50 per cent. of their cases; in Graziani's report, 34 per cent. showed an abolished reflex, 48 per cent. a normal and 18 per cent. an inverted reflex. On the other hand, if we take the analysis of Gorriti,[26] based on a much larger number (207 cases), the results are entirely different: 84 per cent. had a normal, 8 per cent. an inverted and 8 per cent. an abolished reflex. Evidently in any variability curve, gaps are filled when a larger number of cases is studied. The report of Aguglia [17] on seventy-two insane cases, gave 60.8 per cent. as showing an exaggerated reflex, while the report of Gorriti, based on 721 mental cases, showed that the oculocardiac reflex was normal in 81 per cent., inverted in 10 per cent. and abolished in 8 per cent. The reasons for such discordant results are to be found, aside from the individual differences, in the diversity of methods and scales employed.

ORIGINAL INVESTIGATIONS

I undertook a series of observations on different groups of subjects, normal and pathologic, for the purpose of comparing groups instead of individuals. The pathologic cases included tabes, general paresis, psychoneuroses, thyroid states, feeblemindedness and a group of different organic nervous diseases. Most of these patients were taken from the Neurologic Institute, a few from the neurologic department of the Post-Graduate Medical School and Hospital, the feebleminded from Randall's Island, through the courtesy of the medical director, Dr. Vavasour. Seventy-five of the 165 normal subjects were students in the psychologic department of Columbia University, the other ninety were taken at random regardless of age, sex and occupation.

The technic for the induction of the oculocardiac reflex is simple. The subject is placed in the recumbent posture or sits comfortably in an armchair, the head lying on the back of the chair. He is allowed to rest until the pulse becomes quite regular and equal for the four quarters of a minute. Then with the thumb and index or middle finger of the right hand gentle pressure is exerted on the eyeballs through the closed lids. The experimenter stands at the subject's right, taking the pulse at the right wrist. In order to obtain better results, especially when the oculocardiac has to be taken at different times, it is advisable to use an instrument similar to one which I shall describe later.

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25. Graziani, A.: Contributo allo Studio del sistema endocrino simpatico in alcune psicosi, Riv. ital. di neuropatol., psichiat. ed elettroter. **12**: Nos. 2 and 3 (Feb.-March) 1919.

26. Gorriti, F.: El reflejo oculo-cardíaco en 721 enfermos mentales, Semana méd. **23**:671 (Dec. 28) 1916.

In taking the oculocardiac reflex it is better to divide the pulsations into four periods of 15 seconds each. In this way one can easily stop whenever tenderness, or increased sensitiveness or a sharp fall of blood pressure appears and at the same time obtain the average for a minute. This method also gives a measure of the regularity of the oculocardiac reflex. Tabetic patients show no or slight variability in the four quarters of a minute whereas psychoneurotics show the greatest fluctuations; for instance, a psychoneurotic gives this response: pulse: 25, 26, 23, 24, total 98; oculocardiac reflex: 23, 26, 28, 27, total 104. A tabetic patient will give this response: pulse: 22, 22, 22, 22, total 88; or 22, 22, 22, 23, total 89; oculocardiac reflex, 22, 22, 22, 22, total 88; or 23, 22, 22, 22; total 89.

The existence of such great variations in the same person at different times shows the fallacy of designating the reflex by such adjectives as normal, normal feeble, normal medium, normal strong, exaggerated, inverted, when this designation is intended for diagnostic purposes. Moreover, the term "inverted" includes in the same category persons having an oculocardiac reflex from — 1 to — 20 or more; and the term "exaggerated" includes an oculocardiac reflex from + 13 upward. Undoubtedly, the significance—physiologic, pathologic or psychologic—of an oculocardiac reflex of — 1 and — 20 or + 13 and + 60 must be quite different. Therefore, I suggest that the difference in one minute between the pulse rate without ocular pressure and the pulse rate with pressure be always indicated in full with a positive or a negative sign. This algebraic difference should be called reflex index (R. I.). The pulse rate should also be given, as the value of a reflex index is not absolutely the same in a bradycardiac as in a tachycardiac.

Two of the women on whom the experiment was performed—one a neurotic, the other a normal subject—fainted during the experiment; in three other cases the operation had to be stopped because of threatening arrest of the heart. In four epileptic patients pressure of the ocular bulbs caused a seizure.

Five hundred and one subjects were observed, 165 normal and 336 pathologic.

REFLEX INDEX IN NORMAL PERSONS

In Table 1 of my report three groups of normal subjects are reported, a total of 100 persons.

Section A contains fifty university students (male). Of these:

2 or 4 per cent. had a reflex index of 0
14 or 28 per cent. had a reflex index of 1, 2 (positive and negative)
13 or 26 per cent. had a reflex index of 3, 4, 5 (positive and negative)
12 or 24 per cent. had a reflex index of 6, 7, 8 (positive and negative)
5 or 10 per cent. had a reflex index of 9, 10, 11 (positive and negative)
4 or 8 per cent. had a reflex index of 12 or more (positive and negative)

Section B contains twenty-five students (female). Of these:

1 or 4 per cent. had a reflex index of 0
3 or 12 per cent. had a reflex index of 1, 2 (positive and negative)
9 or 36 per cent. had a reflex index of 3, 4, 5 (positive and negative)
8 or 32 per cent. had a reflex index of 6, 7, 8 (positive and negative)
2 or 8 per cent. had a reflex index of 9, 10, 11 (positive and negative)
2 or 8 per cent. had a reflex index of 12 and more (positive and negative)

Section C contains twenty-five male adults taken at random. Of these:

2 or 8 per cent. had a reflex index of 0
5 or 20 per cent. had a reflex index of 1, 2 (positive and negative)
7 or 28 per cent. had a reflex index of 3, 4, 5 (positive and negative)
6 or 24 per cent. had a reflex index of 6, 7, 8 (positive and negative)
1 or 4 per cent. had a reflex index of 9, 10, 11 (positive and negative)
4 or 16 per cent. had a reflex index of 12 or more (positive and negative)

If we were to classify the oculocardiac reflex as normal when the reflex index is from + 5 to + 12, as abolished when the index is from 0 to + 4, as inverted when the index is negative, and as exaggerated when the index is above + 12, the whole group of these 100 normal subjects would show:

In 35 per cent. an abolished reflex
In 33 per cent. a normal reflex
In 9 per cent. an exaggerated reflex
In 23 per cent. an inverted reflex

This group would look quite pathologic to those who expect to find an index of from + 5 to + 12 in normal subjects. Even more pathologic would appear the normal subjects of Table 2, if the same criteria were applied for classification of the oculocardiac reflex. In fact, we would have found the first time:

26 or 40.0 per cent. with an abolished reflex
13 or 20.0 per cent. with a normal reflex
9 or 13.8 per cent. with an exaggerated reflex
17 or 26.1 per cent. with an inverted reflex

And the second time:

36 or 55.6 per cent. with an abolished reflex
9 or 13.8 per cent. with a normal reflex
4 or 6.1 per cent. with an exaggerated reflex
16 or 24.6 per cent. with an inverted reflex

These figures give sufficient ground for rejecting any classification of the reflex index of the kinds that have been so widely accepted. Continuing in the analysis of Table 2, which includes sixty-five normal persons taken at random, whose oculocardiac reflexes were tested two or more times after an interval of from fifteen to ninety days, we found in the first test:

6 or 9.2% with a reflex index of 0
15 or 23.1% with a reflex index of 1, 2 (negative or positive)
17 or 26.1% with a reflex index of 3, 4, 5 (negative or positive)
13 or 20.0% with a reflex index of 6, 7, 8 (negative or positive)
3 or 4.6% with a reflex index of 9, 10, 11 (negative or positive)
11 or 16.9% with a reflex index of 12 and more (negative or positive)

In the second test:

11 or 16.9% with a reflex index of 0
22 or 33.8% with a reflex index of 1, 2 (positive and negative)
14 or 21.5% with a reflex index of 3, 4, 5 (positive and negative)
10 or 15.4% with a reflex index of 6, 7, 8 (positive and negative)
3 or 4.6% with a reflex index of 9, 10, 11 (positive and negative)
5 or 7.7% with a reflex index of 12 and more (positive and negative)

and in the third test, given to thirty persons, we find:

4 or 13.3% with a reflex index of 0
12 or 40.0% with a reflex index of 1, 2 (positive and negative)
9 or 30.0% with a reflex index of 3, 4, 5 (positive and negative)
3 or 10.0% with a reflex index of 6, 7, 8 (positive and negative)
0 or 0.0% with a reflex index of 9, 10, 11
2 or 6.6% with a reflex index of 12 and more (positive and negative)

Again in the first test we found:

6 or 9.2% with a reflex index of 0
42 or 64.6% with a positive reflex index
17 or 26.1% with a negative reflex index

and in the second test:

11 or 16.9% with a reflex index of 0
38 or 58.4% with a positive reflex index
16 or 24.6% with a negative reflex index

In regard to the variations found in the same person in Table 2:

9 or 13.8 per cent. kept the same sign
8 or 11.2 per cent. showed a difference from -1 to $+1$
3 or 4.6 per cent. showed a difference from -2 to $+2$
3 or 4.6 per cent. showed a difference from -3 to $+3$
6 or 9.2 per cent. showed a difference from -4 to $+4$
6 or 9.2 per cent. showed a difference from -5 to $+5$
2 or 3.1 per cent. showed a difference from -6 to $+6$
3 or 4.6 per cent. showed a difference from -7 to $+7$
25 or 38.5 per cent. showed a difference from ± 8 and more

The average index of the following subjects, who were tested from four to six times, gives an idea of the variability of a single index from the average.

In Case 3, 6 tests yielded an average index of 4.83
In Case 7, 4 tests yielded an average index of 2.5
In Case 13, 4 tests yielded an average index of 5.5
In Case 14, 4 tests yielded an average index of 11.25
In Case 15, 6 tests yielded an average index of 1.1
In Case 19, 4 tests yielded an average index of 2.25
In Case 26, 4 tests yielded an average index of —1.0
In Case 28, 4 tests yielded an average index of —0.5
In Case 29, 4 tests yielded an average index of 1.75
In Case 30, 4 tests yielded an average index of 4.5
In Case 44, 5 tests yielded an average index of 1.2
In Case 45, 6 tests yielded an average index of 1.0
In Case 50, 4 tests yielded an average index of —4.5
In Case 56, 4 tests yielded an average index of 4.5

This demonstrates how important it is to have the averages obtained from many trials.

In the absence of the ordinary statistic treatment of the tables reported, a measure of the number of times that each reflex index appeared amongst the normal subjects examined seems necessary before closing the report on Tables 1 and 2.

In the following tabulated summary the reflex indexes are given from 1 to more than 12 with their frequency as encountered in Tables 1 and 2 (first and second trials separately) and finally the frequency in all 230 tests.

It appears from this that the positive indexes are encountered about three times more frequently than the negative ones, and that the small indexes, 1 to 4 and 0, are the most frequent. The positive indexes from 1 to 4 and 0 indexes in the 230 tests constitute 41.3 per cent. of the whole group. This fact demonstrates once more that the so-called abolished reflex (reflex index from 0 to + 4), is more frequently encountered in normal persons and therefore it cannot be considered pathologic.

REFLEX INDEXES FROM 1 TO MORE THAN 12, WITH THEIR FREQUENCY AS ENCOUNTERED IN TABLES 1 AND 2 AND THE FREQUENCY OF THE TWO HUNDRED AND THIRTY TESTS

R. I.	Table 1 5		Table 2 (First Test) 6		Table 2 (Second Test) 11		Total of 230 Tests 22	
	Positive	Negative	Positive	Negative	Positive	Negative	Positive	Negative
1	6	4	7	3	6	5	19	12
2	8	4	3	2	9	2	20	8
3	4	1	4	1	6	2	14	4
4	12	1	5	5	3	1	20	7
5	9	2	2	0	2	0	13	2
6	6	3	6	1	4	4	16	8
7	3	1	2	2	1	1	6	4
8	9	4	0	2	0	0	9	6
9	1	0	0	0	0	0	1	0
10	5	1	1	1	3	0	9	2
11	1	0	1	0	0	0	2	0
12	0	2	1	0	0	0	1	2
13 and more	8	0	10	0	4	1	22	1
Total	72	23	42	17	38	16	152	56

REFLEX INDEX IN PSYCHONEUROTIC AND FEEBLEMINDED PERSONS

A contrast of the findings of the normal subjects of Tables 1 and 2 with those Tables 3 and 4, which include, respectively, psychoneurotic and feebleminded persons, does not reveal much.

In Table 3:

7 or 9.3 per cent. had a reflex index of 0
17 or 22.6 per cent. had a reflex index of 1, 2, positive or negative
16 or 21.3 per cent. had a reflex index of 3, 4, 5, positive or negative
17 or 22.6 per cent. had a reflex index of 6, 7, 8, positive or negative
7 or 9.3 per cent. had a reflex index of 9, 10, 11, positive or negative
11 or 14.6 per cent. had a reflex index of 12 and more

In Table 4:

3 or 6 per cent. had a reflex index of 0
12 or 24 per cent. had a reflex index of 1, 2, positive or negative
15 or 30 per cent. had a reflex index of 3, 4, 5, positive or negative
7 or 14 per cent. had a reflex index of 6, 7, 8, positive or negative
8 or 16 per cent. had a reflex index of 9, 10, 11, positive or negative
5 or 10 per cent. had a reflex index of 12 and more

If we classify the subjects of these two groups by the criteria used for the normal subjects, we do not find much difference, or rather these two groups look more normal than groups of Tables 1 and 2.

In fact, in Table 3:

In 27 or 36.0 per cent. the reflex is abolished
In 25 or 33.3 per cent. the reflex is normal
In 5 or 6.7 per cent. the reflex is exaggerated
In 18 or 24.0 per cent. the reflex is inverted

and in Table 4:

In 18 or 36 per cent. the reflex is abolished
In 21 or 42 per cent. the reflex is normal
In 4 or 8 per cent. the reflex is exaggerated
In 7 or 14 per cent. the reflex is inverted

The data of Table 4 do not agree with the report of Aguglia,[17] who found the oculocardiac reflex most exaggerated in phrenasthenics. A difference is found in comparing the normal groups (Tables 1 and 2) with Table 8, which includes patients taken at random from the clinic suffering from organic nervous diseases. There is a larger number of cases with a 0 index in Table 8.

In Table 8 we find:

11 or 22 per cent. with a reflex index of 0
7 or 14 per cent. with a reflex index of 1, 2, positive and negative
15 or 30 per cent. with a reflex index of 3, 4, 5, positive and negative
7 or 14 per cent. with a reflex index of 6, 7, 8, positive and negative
6 or 12 per cent. with a reflex index of 9, 10, 11, positive and negative
4 or 8 per cent. with a reflex index of 12 and more, positive and negative

However, it must be remembered that among the organic cases also the reflex index is inconstant.

If these cases are classified into abolished, normal, exaggerated and inverted, as has been done with other groups, their distribution will not differ, to any sensible extent, from that found in classifying the results in sixty-five normal persons taken at random.

Here we have:

26 or 52 per cent. with an abolished reflex
13 or 26 per cent. with a normal reflex
4 or 8 per cent. with an exaggerated reflex
7 or 14 per cent. with an inverted reflex

REFLEX INDEX IN THYROID DISEASE

Omitting the cases of exophthalmic goiter in which the secretory alterations are qualitative rather than quantitative, the remainder of this group is constituted of thirty-four hyperthyroid and twenty hypothyroid cases.

Of the hyperthyroid cases

3 or 8.8 per cent. had a reflex index of 0
10 or 29.4 per cent. had a reflex index of 1, 2, positive and negative
4 or 11.7 per cent. had a reflex index of 3, 4, 5, positive and negative
8 or 23.5 per cent. had a reflex index of 6, 7, 8, positive and negative
0 or 0.0 per cent. had a reflex index of 9, 10, 11, positive and negative
9 or 26.5 per cent. had a reflex index of 12 or more, positive and negative
20 or 58.9 per cent. had a negative or 0 reflex index
14 or 41.1 per cent. had a positive reflex index

Of the hypothyroid cases

0 or 0 per cent. had a reflex index of 0
1 or 5 per cent. had a reflex index of 1, 2, positive or negative
4 or 20 per cent. had a reflex index of 3, 4, 5, positive or negative
2 or 10 per cent. had a reflex index of 6, 7, 8, positive or negative
4 or 20 per cent. had a reflex index of 9, 10, 11, positive or negative
9 or 45 per cent. had a reflex index of 12 or more
2 or 10 per cent. had a negative or 0 reflex index
18 or 90 per cent. had a positive reflex index

In this table and in Tables 6 and 7, in which more than one reflex index are given for one subject, the first was computed.

It was observed in many instances that the same subject showed a positive and a negative or 0 index at different times. Although a sharp distinction of the hyperthyroid and hypothyroid cases in sympathicotonic and vagotonic patient (Eppinger and Hess [27]) cannot be drawn, it clearly appears from this table that the hyperthyroid patients tend to react as sympathicotonic and the hypothyroid patients as vagotonic. This fact may help somewhat in the diagnosis of the thyroid states. My results agree with those of Petzetakis,[28] Blanc,[29] and Garnier and Lévi-Franckel.[30] In several hypothyroid patients when large

27. Eppinger, H., and Hess, L.: Pathologie des vegetativen Nervensystems, Ztschr. f. klin. Med. **68**: 1919; Die Vagototonie, Eine klinische Studie, Berlin, 1910.

28. Petzetakis: Le reflexe oculo-cardiaque dans le syndrome hypothiroïdien, Presse méd. **25**:12 (Jan. 8) 1917.

29. Blanc, J.: La dysthroïdie facteur de névrose. Le réflexe oculo-cardiaque régulateur de l'opethérapie thyrodienne, Progès méd. **32**:95 (March 24) 1917.

30. Garnier and Lévi-Franckel: Modifications du réflexe oculocardiaque sous l'influence de la gestation, Bull. et mém. Soc. méd. d. hôp. de Par. **37**:252 (July 24) 1914; Le réflexe oculo-cardiaque dans la grossesse, Compt. rend Soc. de biol. **76**:645 (April) 1914.

doses of thyroid extract were administered, the reflex index became smaller, sometimes even negative.

REFLEX INDEX IN EPILEPSY

On the patient in Case 47, suffering from jacksonian epilepsy, craniectomy was performed. Previous to the operation the reflex was 107—73 = + 34. Eleven months later the patient had a relapse, this time showing an epileptic equivalent. At this time the reflex index was + 16.

Case 46 was also a case of jacksonian epilepsy; the patient was operated on. The two reflexes were taken five days apart, before and after the operation.

The assumption that bromids check the exaggeration of the oculocardiac reflex has not been proved in my cases. Subjects such as Case 11 showed a reflex index of + 19 when taking bromids and + 9 when not taking them; the patient in Case 2 had a reflex index + 9 when taking bromids and + 3 and — 1 when not taking them; the reflex index of the patients in Cases 25, 27 and 50, who were taking bromids, were respectively + 13, + 31, + 13, while those of the patients in Cases 3, 17, 18, 20, 22 and 28, who were not taking bromids, were 0. — 5; — 2, — 1, — 4; — 2; — 1; 0 and 0. The patient in Case 43 had a reflex index of — 4 when taking bromids and — 7 when not taking them. In Case 1 the reflex index was negative with and without bromids. Here also great variations were found in the reflex index when the oculocardiac reflex was taken at different dates. In general, it must be admitted that, despite the variations and the individual differences, the epileptic patients tend to react with a large positive index. The fact that not all epileptic patients are vagotonic demonstrates once more that epilepsy as a clinical entity does not exist; the convulsive seizure is the expression of entirely different pathologic conditions and etiologic factors.

In classifying the subjects of Table 6 we find:

4 or 8 per cent. with a reflex index of 0
10 or 20 per cent. with a reflex index of 1, 2, positive or negative
10 or 20 per cent. with a reflex index of 3, 4, 5, positive or negative
6 or 12 per cent. with a reflex index of 6, 7, 8, positive or negative
4 or 8 per cent. with a reflex index of 9, 10, 11, positive or negative
16 or 32 per cent. with a reflex of 12 or more, positive or negative

The average indexes obtained from the algebraic sum of the reflex index of this and of the other tables show that the epileptic and the hypothyroid patients have the largest average index, namely, + 7.48 and + 11.95, respectively. The smallest average index was found among the tabetic patients (+ 1.23).

REFLEX INDEX IN TABES AND GENERAL PARESIS

Thirty cases of tabes and twenty cases of general paresis are reported in Table 7. What differentiates these subjects, especially the tabetic patients, from all the others, is the small index, even when the oculocardiac reflex is repeated.

Of thirty tabetic patients, we find that in

8 or 26.6 per cent. the reflex index was 0
13 or 43.4 per cent. the reflex index was 1
5 or 16.6 per cent. the reflex index was 2
1 or 3.3 per cent. the reflex index was 3
3 or 10.0 per cent. the reflex index was 4 or more

And of twenty general paresis patients, we find that in

3 or 15 per cent. the reflex index was 0
6 or 30 per cent. the reflex index was 1
6 or 30 per cent. the reflex index was 2
1 or 5 per cent. the reflex index was 3
4 or 20 per cent. the reflex index was 4 or more
20 or 66.6 per cent. of the tabetic patients had a positive index
2 or 6.6 per cent. of the tabetic patients had a negative index
11 or 55.0 per cent. of the general paresis patients had a positive index
6 or 30.0 per cent. of the general paresis patients had a negative index

When the reflex was taken twice or more, the index did not constantly keep the same sign. Only in one case (general paresis) was the index greater than 12.

The absence of oculocardiac reflex in tabes and cerebrospinal syphilis has been attributed to bulbar lesions; to early involvement of the anastomotic fibers running from the fifth to the tenth cranial nerves in the medulla oblongata which have been described by Van Gehuchten and by Ramón y Cajal. If this were true, then we should find bulbar symptoms in all the cases of cerebrospinal syphilis showing a 0 or small index, because we could not imagine a lesion of the anastomotic fibers from the fifth to the tenth cranial nerves which would not sooner or later involve the surrounding fibers. Moreover, in cases of bulbar lesions the oculocardiac reflex has not been shown to be absent, as Guillain and Dubois[31] have pointed out. These authors think that the oculocardiac reflex may serve to differentiate bulbar paralysis from the pseudobulbar. In several cases of bulbar paralysis that I have observed the oculocardiac reflex was not found absent all the time. Of course it will be absent when the center of the reflex arc is involved. It seems more probable that only in a small proportion of the cases of tabes, general paresis and cerebro-

31. Guillain, G., and Dubois, J.: L'abolition et l'inversion du réflexe oculocardiaque dans les paralysies pseudo-bulbaires, Bull. et mém. Soc. méd. d. hôp. de Par. **37**:584 (March) 1914.

spinal syphilis the absence of the oculocardiac reflex is due to a reflex lesion in the center; most cases of so-called absence of the oculocardiac reflex are due to peripheral lesions, when they are not due to constitutional factors.

Undoubtedly the syphilitic virus exerts a selectivs action on both subdivisions of the autonomic system, and when the receptors or the effectors are involved in the lesion, the reflex cannot take place or it will take place in an altered manner. The early digestive, sexual, secretory, and circulatory disorders, the changes in moods and in the affective and emotional life, which are observed in tabes, general paresis and cerebrospinal syphilis before the gross organic signs appear, are indications of the early involvement of the autonomic system. When the autonomic system is functionally affected, as in psychoneurotic patients, we find also cenesthetic, and similar functional signs suggestive of the unharmonious correlation between the two subdivisions of the autonomic system, such as myosis, enophthalmos and reduction of the palpebral fissures in some cases; mydriasis, exophthalmos and widening of the palpebral fissures in others. The conclusion which can be drawn from this consideration is that the oculocardiac reflex, when accurately studied in each person, may give useful information on the condition of the autonomic system and may thus help in the study of the personality.

It is obvious that from the clinical standpoint the oculocardiac reflex cannot constitute a positive diagnostic sign, which could compete with the other well defined and constant symptoms and laboratory findings of tabes. In the early stages of tabes, general paresis and cerebrospinal syphilis, the oculocardiac reflex could serve for differential diagnosis from functional nervous diseases, but the frequency with which the 0 or the small reflex index is encountered in normal persons renders this sign doubtful and unreliable.

FACTORS RESPONSIBLE FOR INDIVIDUAL DIFFERENCES AND VARIATIONS

The factors responsible for the individual differences and variations in the oculocardiac reflex are: (1) age, (2) sex, (3) position of the subject during the experiment, (4) amount of pressure exerted on the eyeballs, (5) physiologic state, (6) physical condition, (7) psychic conditions, and (8) internal or constitutional factors. By internal factors is meant mainly the makeup of the sympathetic endocrine system, which differs greatly among individuals. Broadly speaking, this system forms a large part of what is included in the old terminology of constitution, character and temperament. Besides its relations to other important vital functions, this system is at the basis of cenesthesia. The physical condition is indicated by the development, nutrition,

strength, etc., and in general by the state of health of the subject. The physiologic state includes such functions as digestion, fatigue, the action of drugs, etc., and menstruation or pregnancy in women. The other items do not require explanation.

Factors 1, 2 and 3 can be equalized easily.

Factor 4, which seemed to play a great rôle in the differences and variations found in the same person or in different persons, was eliminated with a simple device consisting of a small instrument resembling a lever having the fulcrum at the center and the power at the ends. The fulcrum was laid on the nose of the subject like spectacles whose lenses were substituted by two light wooden spoons protected with cotton, which could be moved toward the eyeballs with a gentle pressure. The pressure was applied with an ordinary sphygmomanometer having a long bag and a long band so that it could be wrapped around the head of the subject, covering the eyes. The reading in millimeters of mercury indicated the amount of pressure that was used for all subjects submitted to different tests. This method eliminated the errors that may occur when pressure is applied with the fingers. Roubinovitch [32] has devised a similar apparatus, but he did not use the sphygmomanometer reading.

Factors 5 and 6 can be roughly equalized if a careful selection is made of subjects homogeneous as regards their physical and physiologic state.

Factors 7 and 8 cannot be equalized. Here the oculocardiac reflex may be of use. It may constitute an index of the psychic condition and of the sympathetic-endocrine makeup of the subjects, but only when the other factors have been standardized. In normal persons the reflex index may serve as an indication of reflex control, moods, courage, emotions, etc., traits whose scales are wanting. The different behavior of persons when confronted with the same situation is well known. Vague terms, such as temperament, character and neurotic constitution are used to explain why some persons are so abnormally self-conscious as to reach the degree of erythrophobia, why they cannot speak in public without showing a flushed face and having tachycardia, why they cannot undertake or meet a dangerous situation without trembling; whereas others never blush, meet crowds, without any

32. Roubinovitch, J., and de la Sourdière, Regnauld: Le réflexe oculocardiaque dans les hémiplégies et les diplegies cérebrales, Bull et mém. Soc. méd. d. hôp. de Par. **37**:909, 1914; Compresseur oculaire pour la recherche du réflere oculocardiaque, Comp. rend Acad. d. sc. Par. **163**:137, 1916; Progrès méd. **35**: 367 (Aug. 21) 1920. In the latter article the author describes a manometric oculocompressor which is a modification of the one he originated in 1916. When my paper was submitted for publication I did not know that such a modification was being meditated.

sign of anxiety and brave dangers without trembling. The answers to these questions have not yet been given, but it can be said without fear of contradiction that many doubts will be cleared up when a better knowledge of the autonomic system is obtained. For the study of this system, the Dagnini-Aschner reflex and probably also the Herring-Kratschmer reflex may give useful information. In order to obtain information of practical value, however, psychologic methods must be employed first on a large number of normal subjects before passing to the abnormal.

In Table 1, height and weight are given. These measurements, together with other minute anthropometric data not given, were taken in order to learn whether any correlation exists between the morphologic type of subject and the oculocardiac reflex. It seemed to the writer that subjects having small positive, or a 0 or a negative index were more intelligent than those who had a larger positive index. An intelligence test (Otis, form A and Alpha) was given to forty persons of group A (Table 1). Realizing the inconstancy of the reflex index, the results of these correlations cannot be taken at face value. When the average of the indexes, based on many tests, are obtained, the correlation between the average index and the intelligence, and between the average index and the morphologic type, will form the subject of another paper.

INFLUENCE OF CRANIAL NERVES ON REFLEX INDEX

Before concluding I shall report what seems to be an experimental and clinical confirmation of the experiments of Aschner on animals. In order to find what cranial nerves have an influence on the oculocardiac reflex, a group of patients presenting unilateral complete peripheral paralysis of cranial nerves was selected. The oculocardiac reflex was tested at different times on one case each of paralysis of the third, fourth, fifth (motor branch) cranial nerves; fifth (sensory branch) sixth, ninth, eleventh and twelfth cranial nerves; on three cases of second and on eight cases of seventh cranial nerve paralysis. No case of eighth and tenth cranial nerve paralysis was available. In a case of diphtheric polyneuritis, in which both vagus nerves were involved, the index was found to be very large. The patient, a woman 27 years of age, showed left facial paralysis, absolute pupillary rigidity, paresis of the muscles of respiration which kept the patient in severe danger of death for several days, bilateral paralysis of the velum palati, disturbance of deglutition, and laryngeal paralysis. The first time the oculocardiac reflex was $112—64 = +48$; three days later it was $130—58 = +72$. As there was danger of arrest of the heart and of respiratory paralysis, the reflex was not repeated again until four weeks later when the patient's recovery was assured. The reflex

index at that time was between + 6 and + 12. Collet and Petzetakis,[33] in five cases of traumatic vagus lesion, found the reflex inverted. In the case referred to in the foregoing it must be supposed that the thoracic or inferior cardiac branches of the vagus nerves were intact, while the cardiac sympathetic branches were affected, causing the reflex to discharge itself through the vagus alone with production of marked bradycardia.

In the cases of second, third, fourth (motor), fifth, sixth, seventh, ninth, eleventh and twelfth nerve paralysis, no substantial differences were found between the reflex of the left eye and the right eye. The difference was never above 2 counts negative or positive.

In three cases of Bernard-Horner syndrome, differences of a few counts between the right and left index were found. Probably the differences would have been larger if the lesion of the cervical sympathetic nerve had been complete.

The most important observation was made on a patient suffering from "tic douloureux," who was operated on by Dr. Taylor at the Neurologic Institute for posterior root resection of the right trigeminal nerve. After the operation the reflex of both eyes was taken four times, once every second day. The pulse rate is given for quarters of a minute. The findings of the first examination were: 27, 27, 27, 27, average 108; oculocardiac reflex, right, 27, 27, 27, 27, average 108; left, 26, 25, 26, 25, average 102. The findings of the second examination were: pulse 18, 17, 17, 17, average 69; oculocardiac reflex, right, 17, 17, 17, 17, average, 68; left, 15, 15, 15, 15, average 60. The findings of the third examination were: pulse 19, 20, 21, 21, average 81; oculocardiac reflex, right, 20, 19, 20, 21, average 80; oculocardiac reflex, right, 20, 19, 20, 21, average 80; left, 18, 18, 18, 18, average 72. The findings of the fourth examination were: pulse, 23, 21, 20, 20, average 84; oculocardiac reflex, right, 22, 21, 21, 20, average 84; left, 20, 18, 18, 19, average 75.

INFLUENCE OF PAIN STIMULATION ON REFLEX INDEX

Pain stimulation with heavy pressure on the mandible, maxilla and eyebrow gave these results in three trials: left mandible, 20, 21, 21, 21, average 83; left maxilla, 21, 21, 21, 22, average, 85; left eyebrow, 20, 20, 21, 21, average 82; right mandible, 20, 20, 21, 21, average 82; right maxilla, 20, 21, 22, 20, average 83; right eyebrow, 20, 21, 21, 21, average, 83.

This experiment was tried in order to exclude the possibility that

33. Collet and Petzetakis: Le réflexe oculo-cardiaque dans les lésions traumatiques des pneumogastriques, Compt. rend. Soc. de biol. **79**:1147 (Dec. 16) 1916.

the slowing down of the pulse rate, obtained by compression of the right eye, was merely a pain response, which could not take place on the right on account of anesthesia. The same experiment was tried on thirty normal subjects (Table 2) and on thirty pathologic subjects, but no changes in the pulse rate similar to that obtained through ocular compression were found. This experiment proves that the oculocardiac reflex is not a response to pain stimulation; while the case of section of the fifth cranial nerve confirms the existence of a real reflex as described by Dagnini and Aschner. In this case the reflex could not be obtained on account of interruption of the centripetal pathway.

SUMMARY

1. The oculocardiac reflex is subject to individual differences and variations, as is the pulse.

2. Since normal persons are subject to the same changes in their oculocardiac reflex that have been found in many pathologic conditions, the oculocardiac reflex cannot constitute a positive sign for differential diagnosis. It may serve only as an indicator of probability.

3. The classification of the oculocardiac reflex into normal, abolished, inverted and exaggerated classes cannot be accepted on account of the extreme inconstancy of the reflex index: the same normal or abnormal subject may present a positive, a negative, and a 0 index at different times, even when the hour, position, and the amount of ocular compression are kept constant.

About 40 per cent. of the normal subjects examined by the writer showed a reflex index of from 0 to + 4.

4. For clinical and psychologic purposes, the algebraic difference between the pulse rate without ocular compression and the pulse rate during the ocular compression should be given instead of such terms as normal, abolished, inverted and exaggerated. The algebraic difference should be termed the index of the oculocardiac reflex.

5. No scale of the oculocardiac reflex can be reliable which is not expressed in terms of averages. No value can be attributed to an index obtained at a single test. To measure a variable fact, such as the oculocardiac reflex, many measurements are necessary. Therefore the reflex index of a normal or pathologic person should be obtained only from the average of several tests taken at different times. Slight deviations of a few counts above or below the average index should not be given a pathologic significance. Large deviations from the reflex index, although likely to be found in normal states and in normal subjects, may indicate abnormal conditions of physiologic or psychologic states and may also be the expression of pathologic states.

6. In psychology the oculocardiac reflex may be of use for the study of some traits the scales of which are lacking, if a large number of cases and proper methods are used.

7. My researches on pathologic cases have shown that in tabes the reflex index is 0 or very small; exceptionally it surpasses three units; it shows slight variations or no variations at all when taken taken at different times.

In general paresis the index tends to remain small, but cases showing a larger positive or negative index are encountered with much more frequency than in tabes.

In the feebleminded no tendency to a large positive index (exaggerated reflex) was found, as reported by some authors. The groups of psychoneurotic persons, feebleminded persons and persons with organic nervous diseases did not show any substantial variations that could not be found in a group of normal subjects.

In epilepsy, although a well defined tendency toward a large positive index (vagotonic reaction) was found, it was not the rule; cases with small positive indexes, with 0 and negative indexes, were quite often encountered. Bromids did not reduce the reflex index. Large variations from positive to negative indexes were found in the same person at different times.

In thyroid states, a definite tendency was found on the part of the hypothyroid patients to react with a positive index and of the hyperthyroid patients to react with a negative index; this tendency was much more definite in hypothyroid patients. Where the reflex could be repeated at different intervals, large variations were found. Administration of thyroid extract to the hypothyroid patient seemed to produce a reduction of the index.

8. In normal and pathologic cases, pressure over different sensitive spots of the body did not induce the changes in the pulse rate that were obtained with ocular compression.

9. Unilateral paralysis of the second, third, fourth, fifth (motor branch), sixth, seventh, ninth, eleventh, and twelfth cranial nerves did not modify the reflex to a substantial extent. Involvement of the vagus nerves greatly influenced the reflex index; involvement of the cervical sympathetic nerve caused a slight alteration of the index. Resection of the sensory branch of the trigeminal nerve produced suppression of the reflex on the side of the lesion, without influencing the reflex index of the other side. These facts substantiate the results of experiments previously reported, namely, that the centripetal pathway of the oculocardiac reflex is constituted exclusively by the sensory branch of the trigeminal nerve and that the centrifugal pathway is constituted mainly by the vagus and partially by the sympathetic nerve.

37 West Fifty-Fourth Street.

PHENOLSULPHONEPHTHALEIN ABSORPTION FROM THE SUBARACHNOID SPACE IN PARESIS AND DEMENTIA PRAECOX

PAUL G. WESTON, M.D.
WARREN, PA.

The study of absorption of dye from the subarachnoid space is not new. The use of phenolsulphonephthalein is, however, of comparatively recent origin. Dandy and Blackfan [1] used it in their studies of hydrocephalus. (The solution used was neutral in reaction and nontoxic. The solution prepared for kidney function tests should not be used because of its alkaline reaction.) In thirty to fifty minutes after the dye had been injected into the subarachnoid space, traces of it were found in the right lymphatic and thoracic ducts whereas the dye appeared in the blood in three and in the urine in six minutes. In two hours from 35 to 60 per cent. of the dye was recovered in the urine. They conclude that the cerebrospinal fluid passes directly into the blood, and the lymph spaces are not concerned with its absorption. Shortly after the dye had been injected into the lumbar subarachnoid space, it was found in the cerebral ventricles.

Mehrtens and West [2] used phenolsulphonephthalein in a series of cases and reported their results in 1917. They injected 1 c.c. of the neutral dye into the lumbar subarachnoid space, washed the dye from the needle with 3 c.c. of previously withdrawn spinal fluid, thus preventing its escape into the muscles along the needle track when the needle was withdrawn; they catheterized the bladder and determined the time of appearance of the dye in the urine. Quantitative estimations of the amount excreted varied greatly, and the authors suggest that the dye may suffer considerable reduction before complete elimination can take place. Some of the spinal fluids showed a reduction of from 10 to 20 per cent., while others showed no reduction at all. They observed that diseases of the central nervous system, especially when involving the meninges, produce a lengthening of the appearance time to as much as sevently minutes in some cases. In syphilis a lengthening of the appearance time may be produced before any other evidence of central nervous system involvement has appeared, and they conclude that at present no definite conclusion can be drawn as to the exact location of the absorbing tissue.

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1. Dandy, W. E., and Blackfan, K. D.: An Experimental and Clinical Study of Internal Hydrocephalus, J. A. M. A. **61**:2216 (Dec. 20) 1913.
2. Mehrtens, H. G., and West, H. F.: Arch. Int Med. **20**:575 (Oct.) 1917.

Incidental to the routine examination of the spinal fluid in a number of cases of paresis and catatonic dementia praecox, observations were made by the writer on the absorption of the phenolsulphonephthalein from the subarachnoid space.

TECHNIC

Patients were placed on their left side with the buttocks and shoulders on the same level. The skin was cocainized; lumbar punctures made between the third and fourth lumbar vertebrae and a stopcock connected with the needle. Three c.c. of fluid were collected under sterilized paraffin oil for the determination of specific gravity, hydrogen-ion concentration, colloidal gold and globulin tests and the Wassermann reaction. A rubber tube 5 cm. in length with adapters and connected with a 30 c.c. Luer syringe barrel was then attached to the stopcock, and 15 c.c. of spinal fluid were allowed to flow into the syringe barrel. The rubber tube was closed by kinking it on itself and then detaching it. A 2 c.c. tuberculin syringe containing 1 c.c. of a sterilized, neutral solution of phenolsulphonephthalein having a specific gravity of 1.0061 was connected with the stopcock and the dye slowly injected. The syringe was disconnected and the rubber tubing leading from the 30 c.c. syringe was attached to the stopcock. The 15 c.c. of previously withdrawn spinal fluid were slowly injected by controlling the pressure on the piston so that two minutes were required to empty the syringe. In this way the dye was thoroughly washed from the needle. Immediately after the needle was withdrawn the patient was placed on his back, the bladder catheterized and the time of appearance of the dye determined by collecting the urine in a test tube containing a few drops of 10 per cent. sodium hydrate solution.

Three or more days after the intraspinal injection, the bladder was catheterized and as soon as it was empty, 1 c.c. of the neutralized dye was injected deep into the deltoid muscle and the time of its appearance in the urine determined. The stopcock made it possible to withdraw fluid and inject the dye without losing a drop of either.

DISTRIBUTION OF THE DYE

It was necessary first to determine the average amount of cerebrospinal fluid in cases of dementia praecox and paresis. Such information was obtained from cadavers. Immediately after death, with the body on its back and the head slightly elevated, a needle was inserted into the cisterna magna and another into the lateral ventricle, a small hole having been bored through the skull for the purpose. The fluid was collected in a graduated cylinder and the head rotated

several times until no more fluid flowed from the cisterna. The body was then turned on its side with shoulders slightly elevated and a needle inserted between the twelfth thoracic and first lumbar vertebrae. The fluid flowing from this needle and representing the amount in the canal from the medulla to the end of the cord was collected and measured. When no more fluid flowed, a fourth needle was inserted between the last lumbar and first sacral vertebrae. Fluid flowing from this needle represented the amount in the lumbar area. The average amount of fluid in sixty dementia praecox cases of all forms was 110 c.c. in the skull, 15 c.c. in the thoracic part of the cord and 15 c.c. in the lumbar area. From twenty-eight cases of paresis the amounts were 135, 18 and 15 c.c., respectively.

TABLE 1.—Maximum and Minimum Amounts of Fluid Found

	Maximum Amount in C.c.			Minimum Amount in C.c.		
	Skull	Thoracic Part of Cord	Lumber Area	Skull	Thoracic Part of Cord	Lumber Area
Dementia praecox	120	18	18	90	13	12
Paresis	150	19	19	110	13	13

The specific gravity of the fluid from the cases observed (determined by pyknometer) varied from 1.0061 to 1.0063 in the dementia praecox group and from 1.0061 to 1.0073 in the paretic group. The specific gravity of the dye was 1.0061. One would therefore expect the dye to diffuse slowly in those cases in which the specific gravity of the fluid was the same as that of the dye solution and to rise to the higher portions of the cord when the dye solution was lighter than the spinal fluid.

A. E. Barker[3] studied frozen sections of cadavers and found that with the body on its back, the highest point in the spinal canal was in the cervical region and next to this, the space between the third and fourth lumbar vertebrae. The canal slopes in both directions from this interspace. The cephalic slope continues downward to the fifth and sixth thoracic vertebrae when it begins to run upward again until the third cervical vertebra, or if the head is on a pillow, until the foramen magnum is reached. It would, therefore, seem that the most appropriate place to inject the dye would be between the third and fourth lumbar vertebrae, for this would give it an opportunity to flow in both directions and cause greater diffusion. One would expect to find the dye as far up as the fifth or sixth thoracic vertebrae in a few minutes. Dandy and Blackfan state that the dye finds its way into the cerebral ventricles a few minutes after it is injected into the lumbar subarachnoid space.

3. Barker, A. E.: Brit. M. J. 1:597, 1912.

In order to determine how far the dye diffused, 1 c.c. was injected as described above into the lumbar subarachnoid space of a cadaver immediately after death. Ten minutes later the cisterna magna was punctured, the buttocks elevated so that the spinal canal was at an angle of about 45 degrees, and 20 c.c. of clear fluid withdrawn before any dye appeared. In another instance puncture between the fifth and sixth thoracic vertebrae with the body placed horizontally yielded clear fluid—no dye was present. These experiments were repeated on other cadavers one hour after injection of the dye, with the same results. The conclusion is that the dye does not leave the lumbar area in cadavers. In a selected number of dementia praecox and paretic cases puncture of the cisterna magna, after injection of the dye into the lumbar subarachnoid space, yielded clear fluid free from traces of color. Punctures were made, one in each case, at intervals of one-fourth, one-half, one, three and five hours after the injection of the dye. In these cases there was no diffusion upward of the dye as far as the cisterna. It, therefore, remained in the lumbar or thoracic regions or was destroyed or absorbed before it could reach the cervical region.

To determine whether the cerebrospinal fluid destroyed the dye, its effects were studied in the test tube. Because of the above findings, it was assumed that all the unabsorbed dye was in the lumbar and lower thoracic regions, and, since there was an average of 20 c.c. of fluid in these areas (15 c.c. in the lumbar area plus one-third of the amount in the whole thoracic area), each cubic centimeter contained approximately one-twentieth c.c. of the dye. This amount of dye was then added to 1 c.c. of freshly drawn fluid collected under sterilized paraffin oil and the same amount was added to 1 c.c. of sodium carbonate solution having a p_H of 7.4, which was the same as that of the spinal fluid. The tubes were covered with sterilized paraffin oil, closed with cork stoppers and placed in the dark at 37 C. for twenty-four hours. At the end of this time one-twentieth c.c. of the dye was added to a freshly prepared sodium carbonate solution having a p_H of 7.4. Two c.c. of 5 per cent. sodium hydrate solution were then added to each tube and their contents compared in the Kober colorimeter. The freshly prepared sodium carbonate solution was used as the standard. There was no determinable destruction of the dye by the spinal fluid in any instance. Mehrtens and West [2] found a reduction of 10 to 20 per cent. in some cases and no reduction at all in others.

The dye was therefore not reduced by the fluid in a test tube under the above conditions and perhaps was not reduced by it in the spinal canal. Quantitative estimations of the amount of dye elimi-

nated were not made. The observations that have been made indicate that the dye is absorbed from the lumbar region and that it does not reach the cisterna in five hours.

APPEARANCE TIME

Dandy and Blackfan found the dye in the urine six minutes after it had been injected into the lumbar subarachnoid space. The tables in the paper of Mehrtens and West give the appearance time as four to ten minutes—in one case fourteen minutes. These figures, of course, are for normal subjects or at least for subjects not having demonstrable involvement of the central nervous system.

TABLE 2.—DEMENTIA PRAECOX CASES

Number	Age	Duration, Years	Appearance Time, Minutes	
			Intraspinal	Intramuscular
9965	30	6	25	15
5092	44	13	25	12
6068	34	15	28	13
8529	27	6	30	20
8278	29	7	30	11
8477	34	10	30	15
4000	51	25	30	10
5548	38	15	35	6
8672	27	7	37	18
5220	44	20	37	10
10131	27	3	37	12
7938	35	15	40	7
6209	37	19	41	10
6386	39	20	42	7
6900	30	11	45	4
8229	28	8	45	11
6043	33	5	48	6
7331	34	12	50	10
9650	26	3	53	9
9056	26	5	55	13
8916	28	5	58	10
5145	42	16	60	11
8022	24	6	65	17
5975	33	13	80	11
7516	30	10	90	7
5945	33	14	96	10
7908	30	10	100	8
10564	19	1	104	10
		PARESIS CASES		
10150	37	4	12	18
10719	34	1	13	9
10675	33	3	25	15
10716	46	2	25	10
9815	38	4	26	14
10189	34	2	28	18
10508	54	1	28	19
10525	39	2	30	14
10647	39	2	31	10
9550	39	5	32	12
10621	49	1	33	20
10689	39	1	34	12
8296	43	7	35	14
10724	47	2	40	10
10654	52	2	42	12
10065	31	3	51	17
10414	40	4	68	9

From the table it will be seen that the shortest appearance time in the series was twelve minutes. It occurred in a case of paresis. The longest appearance time was in a case of catatonia, 104 minutes. In the paretic cases the time varied from twelve to sixty-eight minutes and in the dementia praecox group from twenty-five to 104 minutes. After intramuscular injection, the appearance time varied from four to twenty minutes.

SUMMARY

One c.c. of neutral, sterilized solution of phenolsulphonephthalein having a specific gravity of 1.0061 was injected into the lumbar subarachnoid space and the contents of the needle washed into the canal with 15 c.c. of previously withdrawn spinal fluid. The time of appearance of the dye in the urine was then determined. Twenty-eight cases of catatonic dementia praecox and seventeen cases of paresis were observed. Age, duration of the psychosis, physical and mental condition, so far as could be determined, had no constant effect on the appearance time. In all cases the dye was longer in making its appearance in the urine than the normal time stated by Dandy and Blackfan, six minutes, and by Mehrtens and West, four to ten minutes. In these observations the time varied from twelve to sixty-eight minutes in the case of paresis and from twenty-five to 104 minutes in the case of catatonic dementia praecox.

The specific gravity of the spinal fluid varied from 1.0061 to 1.0063 in the dementia praecox group and from 1.0061 to 1.0073 in the paretic group. The hydrogen-ion concentration in all cases was 7.4. The Wassermann reaction, gold and globulin tests were all negative in the dementia praecox cases and were positive in the paretics.

The dye was not found in fluid drawn from the cisterna magna at any time up to five hours after it had been injected into the lumbar subarachnoid space. The absorption of the dye took place from the lumbar region.

THE FALSE LOCALIZING SIGNS OF SPINAL CORD TUMOR

CHARLES A. ELSBERG, M.D.
NEW YORK

The development of the surgery of tumors of the spinal cord and membranes has been due to the advances in our knowledge of the fiber tracts and their pathways and of the localization of functions in the different tracts and segments of the cord. With these advances the names of Bechterew, Cajal, Koelliker, Lenhossek and others, and of Brown Séquard, Sherrington, Thorburn, Mackenzie, Allen Starr and Head are indelibly associated.

Although much is still unknown, it is now possible to diagnosticate the level of a spinal new growth, the side of the cord on which the tumor lies, and the relation of the growth to the cord and to the structures which surround it. Thus, in a large number of cases in our experience, it has been possible to determine before the operation whether the tumor was intramedullary or extramedullary; whether it lay in front or behind, to the left or to the right of the cord, and whether it lay behind or in front of one or more nerve roots.

The diagnosis "spinal cord tumor" can be correctly made in the majority of instances, if a careful history of the beginning and progress of the symptoms is obtained, and the examination is a thorough one. It is unusual to find a tumor when none is expected, and it is becoming increasingly rare wrongly to make the diagnosis "spinal cord tumor." We are accustomed to divide the patients into three groups: (1) those in whom the diagnosis can be made with certainty, (2) those in whom a tumor is probable, and (3) those in whom a tumor is possible but not probable. In the patients in the first group a tumor was usually found; in those of the second group, it was often present, and in those of the third group the disease was rarely a new growth.[1]

Regarding the question of the correct level diagnosis, we have had, as reported elsewhere,[2] some patients in whom the signs were wrongly interpreted, or the examination was not made with sufficient care, so that the tumor was first looked for several segments higher or lower than where it was finally found. In other patients, without doubt, the level diagnosis was correct, but the desired segments of

1. In my eighty-one operations for spinal cord tumor, the correct diagnosis was made seventy-three times; in the other eight patients a tumor was considered one of the possibilities.

2. Elsberg, C. A.: Concerning Spinal Cord Tumors and Their Surgical Treatment, Am. J. M. Sc. **159**:194 (Feb.) 1920.

the cord were not at first exposed by the laminectomy, either because the wrong vertebral arches were removed or on account of the natural variations in the relation between the vertebrae and the cord segments. In not a few cases, several more laminae had to be removed before the new growth was exposed. With increasing experience these mistakes have become more rare, but I am conscious that others might have done better than I have done.

In some patients, however, the symptoms and signs noted at repeated examinations by different examiners pointed clearly to a certain level and a definite location, but the growth was found to have entirely different relations to the cord. I have characterized these as cases with "false localizing signs."

CHANGES IN THE SENSORY LEVEL SIGNS OF SPINAL TUMORS

The following histories are given as examples of this phase of our subject:

CASE 1.—*History.*—There were increasing spinal symptoms of sixteen months' duration and level symptoms at the seventh thoracic segment. Laminectomy was performed. No tumor was found. Four years later, level symptoms at the seventh and eighth cervical segment appeared. Laminectomy was performed, and an extramedullary tumor was removed.

Mrs. L. G. was admitted to the New York Neurological Institute as a private patient of Dr. Foster Kennedy in June, 1919. She was 35 years of age, married, and had three healthy children. During a period of fourteen years, she had sprained her right ankle a number of times. In February, 1910, she had an attack of influenza, after which she noticed that her left lower extremity felt heavy and that she frequently had a tingling sensation in the left leg and foot; soon after the right lower extremity became similarly affected. Two months after the onset of her symptoms, the patient began to have decided difficulty in walking; the limbs became progressively weaker during the fall and winter of that year. She wore a brace on the right ankle on the theory that the pain and weakness were due to the repeated sprained ankle. The right leg was always weaker than the left, but the tingling was most marked in the left leg. In the spring of 1911, she began to have pain in the back. She was treated for flat feet, and arch supports were ordered for her; these did not improve the condition. At about this time she first noticed a loss of feeling in her lower limbs. The limbs became more and more stiff and weak and she became bedridden; she had some difficulty with micturition.

First Examination.—June 16, 1911: The patient was well nourished. The cranial nerves were normal; the pupils reacted well to light and accommodation; nystagmus was not present. Upper Extremities: Power was good, the right equal to the left; biceps, triceps and wrist reflexes were active and equal. Sensation was normal in both extremities. Abdominal Reflexes: These could not be obtained (the patient was stout). Lower Extremities: Both lower extremities were spastic and weak, the right much weaker than the left. The patient had greater difficulty in raising the right limb from the bed than the left one. Flexor power at the knee and ankle was also much weaker on the

right side. Knee and ankle reflexes were much exaggerated and double ankle clonus and the Babinski reflex were present. The extension of the large toe was more marked on the right.

Sensory Status (Fig. 1): Sensation in the chest and upper limbs was normal. Posteriorly on the left side, below the level of the ninth thoracic spine there was almost complete loss of tactile, pain and temperature sensibility, while anteriorly the same condition obtained up to the same level. Over the

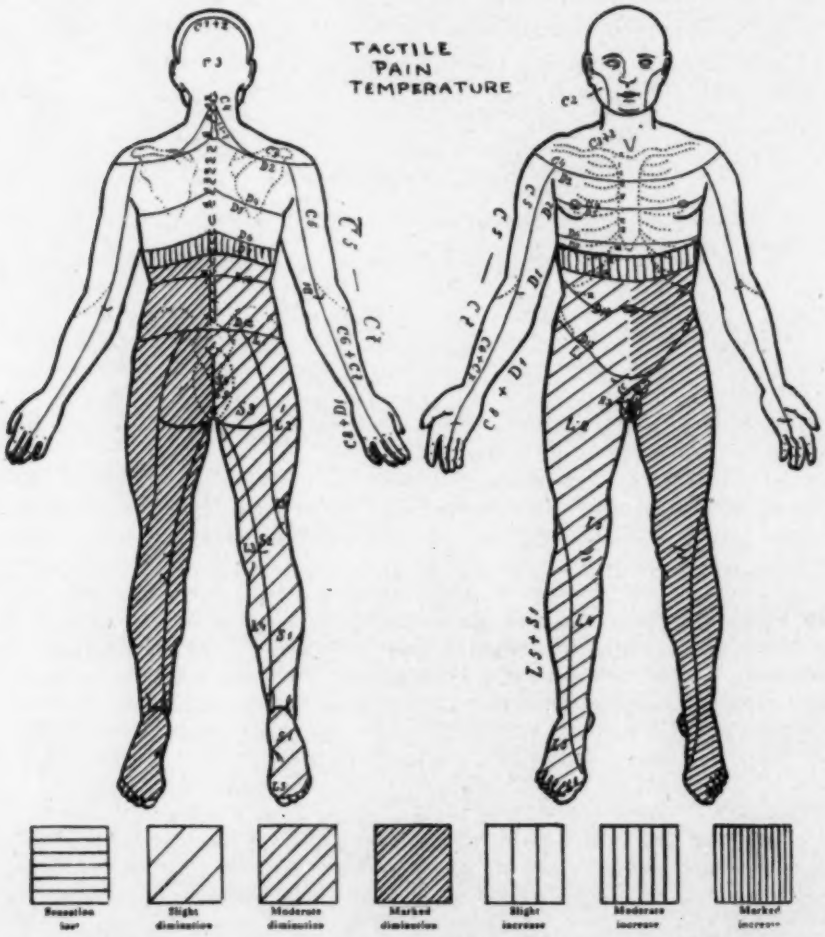

Fig. 1 (Mrs. L. G.).—False sensory localizing signs of a spinal cord tumor at the level of the eighth cervical segment. Compare with Figure 2.

corresponding area on the right side, there was a moderate loss of touch, pain and temperature sensibility. Above the level described there was a band of hyperesthesia for all sensations, about 2 inches wide. Behind, this hyperesthesia extended up to the spine of the eighth dorsal vertebra, which appeared to be tender to pressure. Sense of position of the toes and vibratory sense were completely lost in the right foot and leg; in the left toes, one mistake was made in five tests.

Roentgen-ray examination of the spine was negative. The fluid obtained by lumbar puncture was clear; globulin was not increased; there were no cells; the Wassermann test was negative.

Treatment and Course.—The diagnosis of an extramedullary tumor at or above the seventh thoracic segment was made and a laminectomy (fifth to ninth dorsal) was performed by me on June 26. The operation failed to reveal a tumor even after careful exploration, but it was noted in the record that the posterior spinal vessels were distended with blood and that, after the first escape of cerebrospinal fluid when the dura and arachnoid were incised, there was no further leakage of fluid from above. The patient recovered satisfactorily from the operation and she left the hospital against advice three weeks later. She was seen at her home about three months later. Her condition was unchanged.

Second Admittance to Hospital.—Nothing more was heard from her until March, 1915, about three and one-half years after the operation. She was then admitted into Mt. Sinai Hospital as a private patient of Dr. I. Strauss and was referred to me by Dr. Strauss for operative interference. During the three and one-half years the patient had become progressively worse; she had gradually lost all power in the lower extremities, and the upper limbs—especially that of the right side—had become weaker. She had lost complete control of the bladder, and her bowels had become obstinately constipated.

Examination.—Examination made March 11, 1915, revealed the following: The cranial nerves were normal, except that the right pupil was smaller than the left. Upper Extremities: Movements at the shoulders were good; at the elbows, flexion was good, extension weak, especially on the right side. The biceps reflexes were present and equal; triceps and radius and ulnar reflexes could not be obtained. Abdominal reflexes were not obtainable. There was a marked spastic paraplegia of both lower extremities. The patient was unable to move either limb and on the least irritation, there was a tremendous flexor spasm of both extremities. Knee reflexes were greatly exaggerated. Double ankle clonus, double Babinski and Gordon reflexes were present. There was a complete loss of all sensation—superficial and deep—below the level of the seventh cervical area on both sides (Fig. 2). The symptoms now pointed clearly to a tumor at the seventh to eighth cervical segments, on the posterior surface of the cord and to the right.

Treatment and Course.—At the operation (Dr. Elsberg) on March 15, 1915, the arches of the sixth and seventh cervical and first thoracic vertebrae were removed. When the dura was opened a tumor, about 1 inch long and lying on the posterior surface of the cord slightly more on the right side, was exposed (Fig. 3). The growth, which proved to be an endothelioma, was removed without difficulty, leaving a deep depression in the cord. Convalescence from the operation was uncomplicated, but there was no improvement in the paraplegia.

Case 2.—*History.*—Spinal symptoms had been present for one year. There were symptoms at the eleventh to twelfth thoracic segments. Laminectomy was performed; no tumor was found. Seventeen months later, there were level symptoms at the fifth thoracic segment. Laminectomy was performed and an extramedullary tumor was removed at the fourth thoracic segment. The patient recovered.

H. R., 36 years of age, a patient of Dr. C. L. Dana, was admitted into the New York Neurological Institute in January, 1912. One year before, he first noticed a tickling sensation in the calf of the right leg and an annoying sensation as if the outer side of the leg were "rubbed with sand paper." A few months later he noticed that the lower part of his body was not as sensitive to cold as the upper. Three months later, the right knee had become stiff and he had difficulty in walking. Four months before admittance, the

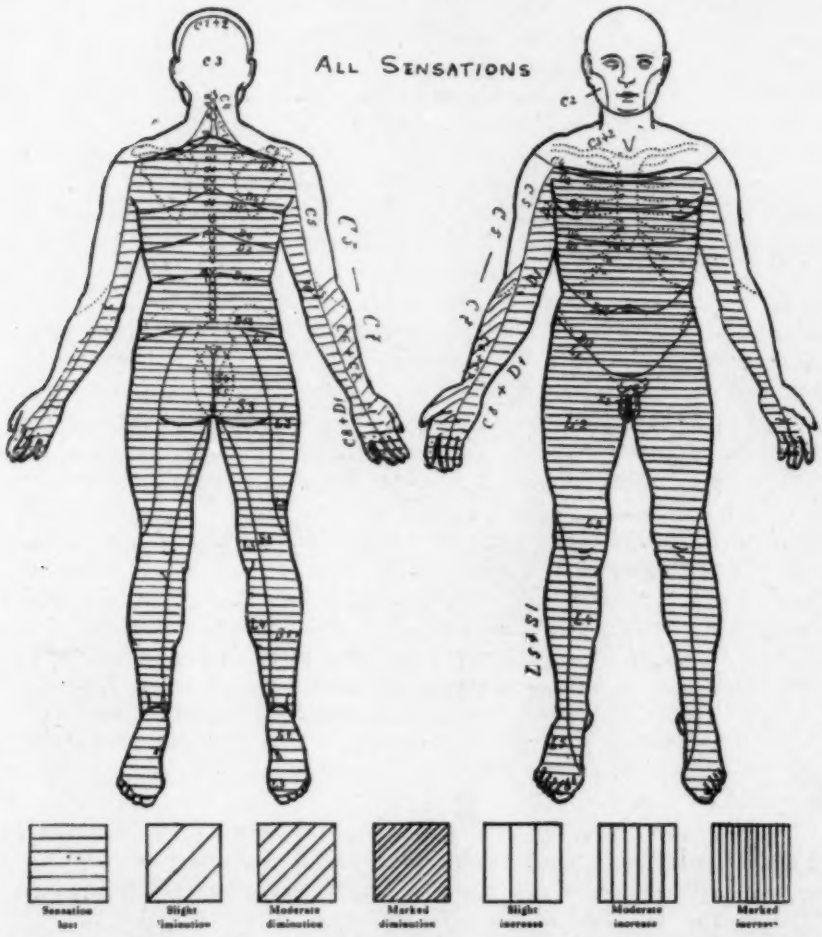

Fig. 2 (Mrs. L. G.).—Sensory disturbances, four years later. Correct sensory level signs of a tumor at the eighth cervical segment. Compare with Figures 1 and 3.

left lower extremity became weak and stiff. The weakness and stiffness of the lower limbs became progressively worse up to the time of admittance. He had no trouble with his upper extremities. For eight months he had increasing difficulty in urination.

Examination.—Jan. 25, 1912: The patient, who was strong and muscularly well developed, walked with great difficulty on account of marked spas-

ticity of both lower extremities. The cranial nerves were normal; nystagmus was not present; the pupils reacted well to light and accommodation. Upper Extremities: Power was good, that of the right equal to that of the left; the reflexes were active, those of the right equal to those of the left. The abdominal reflexes were active, those of the right side equal to those of the left. Cremasteric reflexes were weak on both sides. Lower Extremities: Both were spastic; the knee and ankle reflexes were much exaggerated. Double ankle clonus and the double Babinski reflex were present. There was no disturbance of the articular or vibratory sense. There was no disturbance of tactile sense, the slightest touch with absorbent cotton being felt distinctly over both lower limbs. Heat and cold were not felt below the level of the eleventh thoracic segment; pain sensibility was distinctly diminished over the third and fourth lumbar segments on each side, and slightly diminished on L 2. Just below the umbilicus, there was a band of marked hypalgesia about 2 inches

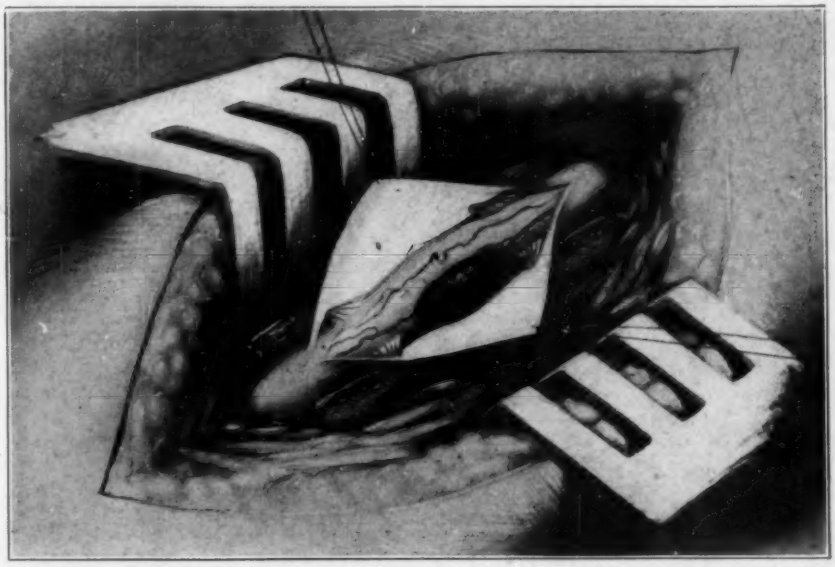

Fig. 3 (Mrs. L. G.).—Extramedullary tumor at the eighth cervical segment. Compare with Figures 1 and 2.

wide, which extended all around the body (Figs. 4 and 5). The Wassermann tests of the blood and cerebrospinal fluid were negative. The roentgen-ray examination was negative.

The patient left the hospital after a few weeks; he returned four months later in a much worse condition. His back had become stiff; he was bedridden. The lower limbs were much more weak and stiff. He was having considerable bladder trouble, and he could not control his bowel movements.

Second Examination.—Physical examination, July 4, 1912, revealed: The upper extremities were normal. The upper abdominal reflexes were present and active; the lower abdominal reflexes were weak. The spasticity of the lower limbs had increased very much, and there was an almost continual clonus of the extensors of the thigh. With great difficulty, the patient could lift his lower limbs a few inches from the bed. Flexor power at the knees was

weak. The knee and ankle reflexes were much exaggerated with double ankle clonus, Babinski and Oppenheim reflexes—a little more marked on the right side. There was no loss of vibratory or articular sense.

The sensory examination revealed: The disturbances extended on both sides up to the level of the eleventh and twelfth thoracic segments, and all sensations were involved, although pain and temperature sense were markedly affected, while tactile sensation was only slightly interfered with.

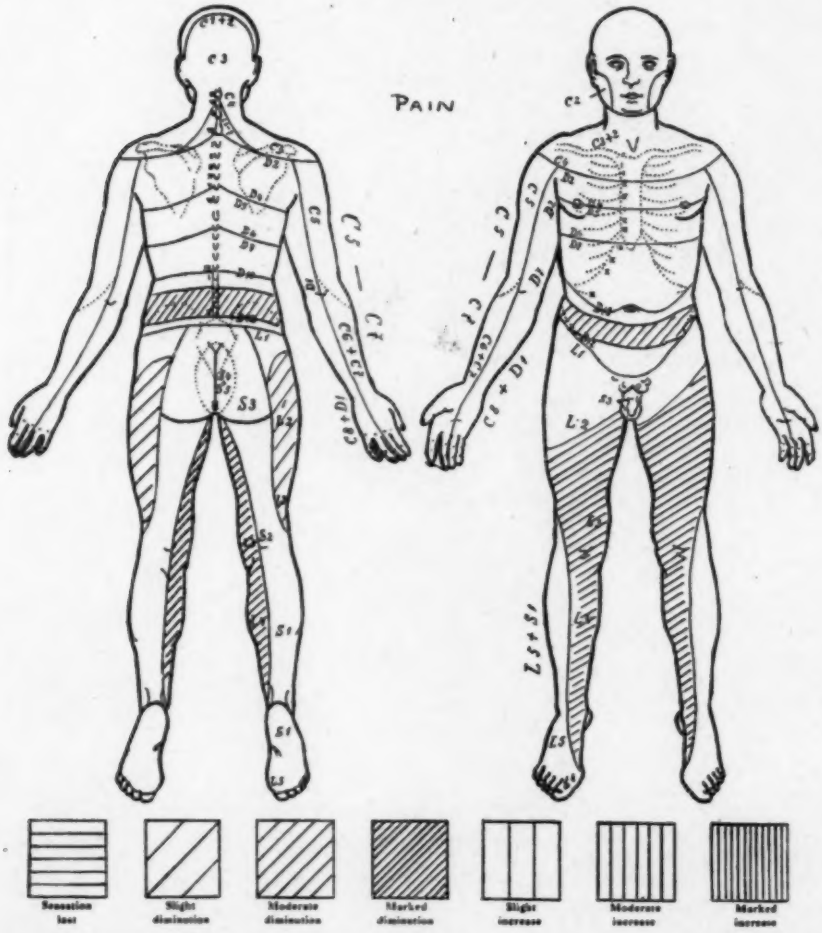

Fig. 4 (H. R., Jan. 25, 1912).—False sensory localizing signs of a spinal cord tumor at the level of the fourth thoracic segment. Compare with Figures 6 and 7.

Treatment and Course.—July 5, 1912, an exploratory laminectomy was performed by me. The arches of the ninth, tenth, eleventh and twelfth thoracic and of the first lumbar vertebrae were removed. When the dura and arachnoid were incised, the cerebrospinal fluid spurted out to a height of 12 inches. There was no evidence of tumor, the probe passed upward and downward in front

and behind the cord without meeting any obstruction. The wound was closed. The patient left the hospital unimproved after one month.

Second Admittance to Hospital.—Sixteen months later, he was seen at the Montefiore Home, where he had been admitted to the service of Dr. S. P. Goodhart, some months before. He had a paraplegia in extension, although with great effort he could flex his knees to about a right angle. The abdom-

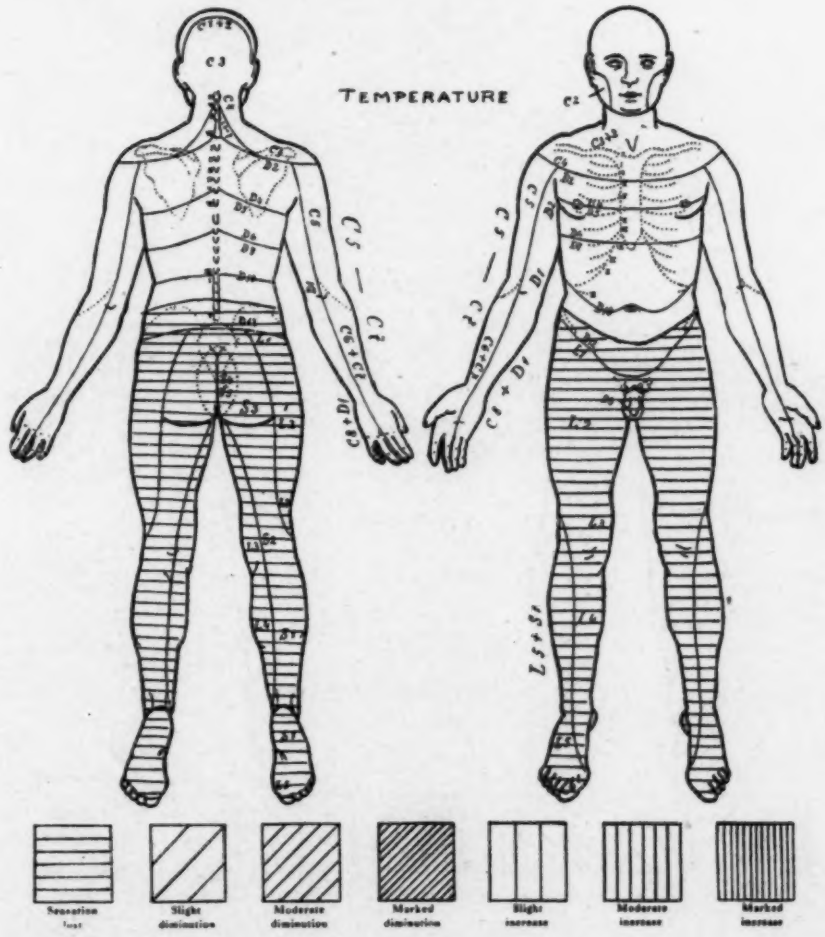

Fig. 5 (H. R., Jan. 25, 1912).—False sensory localizing signs of a spinal cord tumor at the level of the fourth thoracic segment. Compare with Figures 6 and 7.

inal and cremasteric reflexes were present but were weaker on the left. He had markedly exaggerated knee reflexes, double inexhaustible ankle clonus, double Babinski, Chaddock, Mende and Gordon reflexes. There was a well marked loss of articular sense in the left lower limb. The sensory changes (Figs. 6 and 7) extended up to the fifth and sixth thoracic segments.

Treatment and Course.—Jan. 2, 1914: Laminectomy was performed by me. Arches were removed at D 4 and 5, and then arches at D 2 and 3. The extramedullary fibroma which lay behind and on the left side of the cord and pushed the cord over to the right (Fig. 8) was removed. The tumor was easily raised from its bed and removed. It measured 3 by 1.5 cm. The patient improved rapidly and six months after the operation was entirely well.

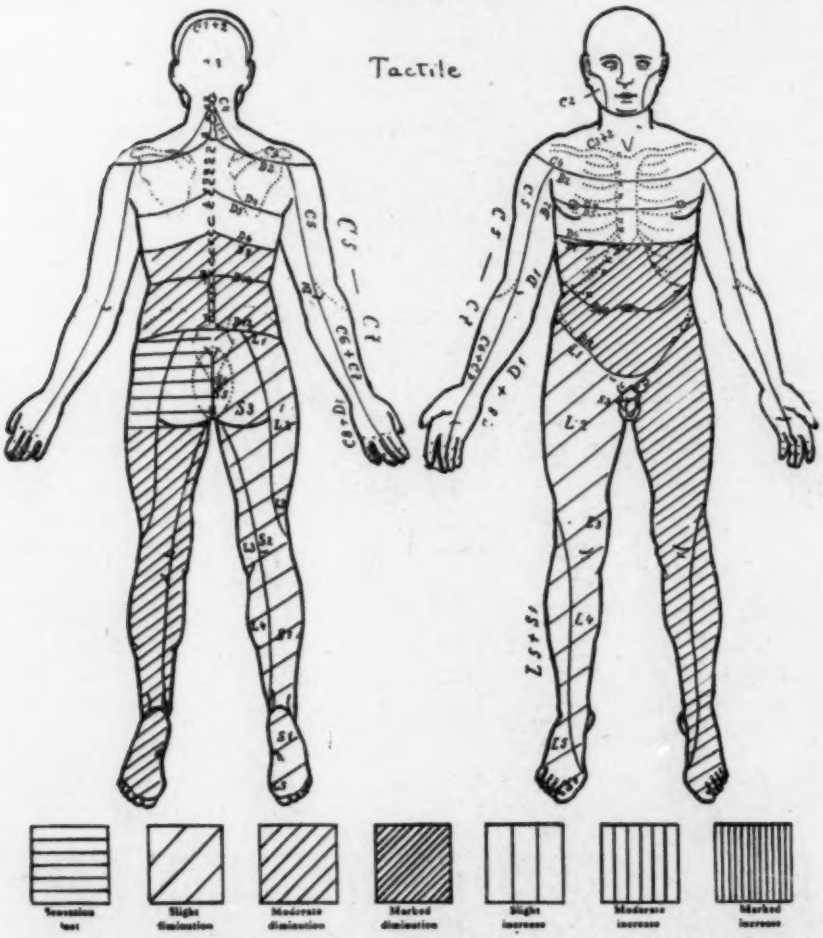

Fig. 6 (H. R., Nov. 29, 1913).—Sensory disturbances seventeen months later than those shown in Figures 4 and 5. Correct sensory signs of a tumor at the fourth to fifth thoracic segments. Compare with Figures 4, 5 and 8.

To summarize: In Case 1 there were at first distinct level signs at the seventh thoracic segment and three and one-half years later, signs at the eighth cervical level. The tumor was removed from the eighth cervical level. In Case 2 there were signs of a tumor at the fourth thoracic level; two years before there were signs and symptoms of a new growth at the eleventh thoracic segment. In each of the

patients, the first laminectomy was performed at a much lower level than that at which the tumor was found at the second operation.

How can these cases be explained? It will be noted that, in both instances, there were no root symptoms, and that in Case 1, the symptoms referable to the upper extremity appeared several years after those referable to the lower limbs.

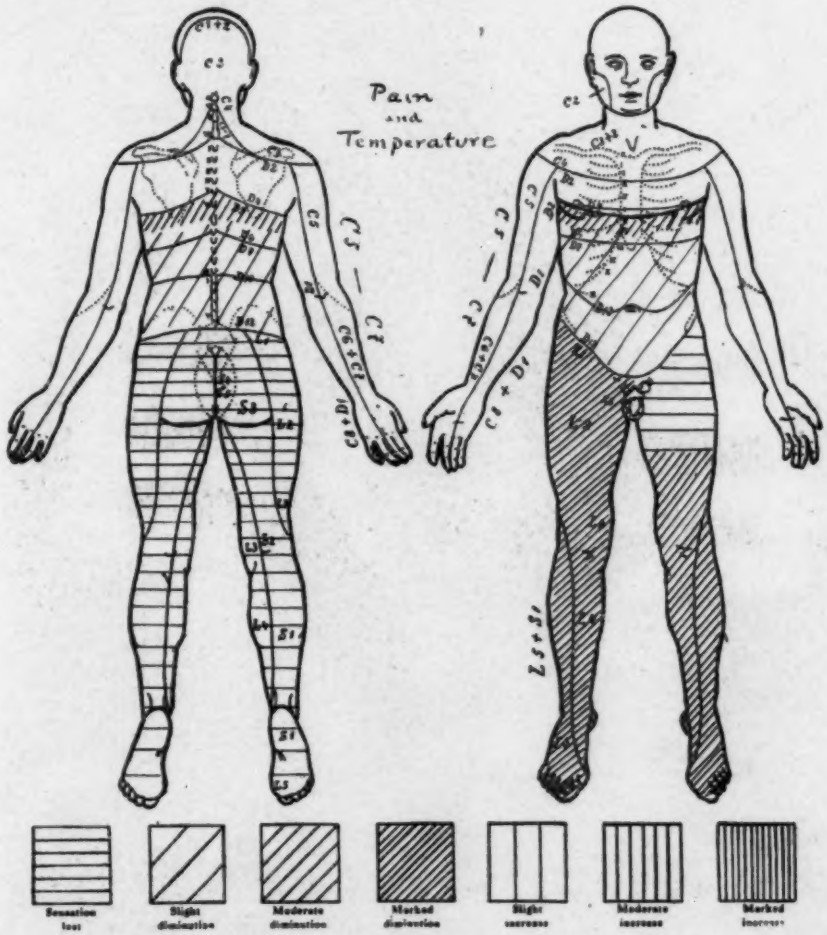

Fig. 7 (H. R., Nov. 29, 1913).—Sensory disturbances seventeen months later than those shown in Figures 4 and 5. Correct sensory signs of a tumor at the fourth to fifth thoracic segments. Compare with Figures 4, 5 and 8.

In looking over my records, I have been impressed by the frequency with which patients with spinal tumors in the cervical region first complain of sensory and motor symptoms referable to the lower extremities. In twenty-five cases of extra- and intramedullary cord tumors in the cervical region, six patients stated that the first symp-

toms they observed were in the lower extremities; in three, weakness, stiffness and sensory disturbances preceded symptoms in the upper limbs by many months, and two of the five patients had no knowledge of any motor or sensory disturbances in their upper extremities when they were first examined.

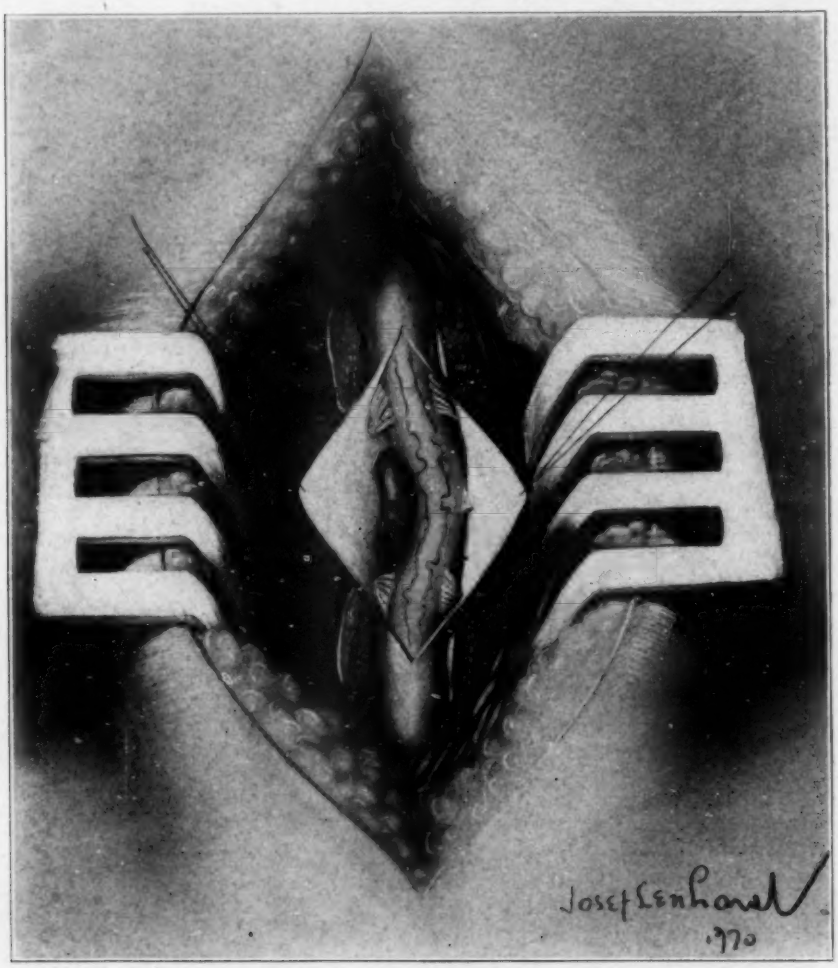

Fig. 8 (H. R.).—Extramedullary tumor at the fourth thoracic segment. Compare with Figures 4, 5, 6 and 7.

When, in an expanding intramedullary or extramedullary spinal lesion, the loss of sensation is not a complete one, the boundary between parts of normal and abnormal sensibility is often uncertain. If the impulses which pass upward have not been completely interrupted at a certain level, the area of disturbed sensation may merge

gradually into parts of normal sensation. This change from disturbed to normal may be so gradual that above the area in which sensibility disturbances can be recognized with the ordinary tests, there may be a broad area (extending over a number of cord segments) in which sensation is apparently normal. As soon as the interruption becomes more marked, however, the sensory disturbances shift to a higher level, which is the true segmentary level.

It is probable that the fibers for the different extremities and even for parts of the extremities are grouped together in the spinal pathways.[3] If there is such a definite grouping of sensory fibers, one can understand that with the gradual crossing of the fibers for pain and temperature, and perhaps also for tactile sensation, only a small part of these fibers may at first be interfered with. The affected fibers may be those that supply areas considerably below the actual level of the lesion, and hence the sensory level obtained early in the course of the disease may be one considerably below the real level of the lesion.[4]

ERRORS IN INTERPRETATION OF SIGNS REFERABLE TO THE SIDE AND PART OF THE CORD AFFECTED

In a paper published some years ago,[5] attention was called to the fact that, in rare instances, a tumor on one side of the cord may dislocate the cord to the opposite side to such an extent that the pressure of the cord against the wall of the spinal canal may cause symptoms referable to the side of the cord opposite to that of the tumor. I have seen two cases of this condition, in which, with ill-defined Brown Séquard symptoms, the greatest motor loss was on the opposite side and the marked sensory disturbances on the same side as the tumor. In both these patients the tumor had been localized on the opposite side to that at which it was found at operation. In a third patient, we were uncertain as to the side of the cord on which the tumor lay, because the marked motor symptoms and signs were on the opposite side, and the distinct sensory signs on the same side as the root symp-

3. Most of the evidence, hitherto adduced, seems to support the opposite point of view, i. e., that motor, and also to a great extent sensory, fibers are not grouped according to the areas they supply. In another paper I hope to give strong evidence that the contrary is true, and that there is a definite grouping of fibers within the sensory and motor pathways of the spinal cord in accordance with the parts of the body or of the extremities supplied.

4. Most writers state that the fibers conveying the sensations of pain and of heat and cold require from two to three segments for their crossing. I believe that Head is correct in his statement that the crossing is much more gradual, requiring from five to six segments.

5. Am. J. M. Sc. **149**:337 (March) 1915.

toms. The condition here described is analogous to what is not so rarely seen in tumors in the cerebellopontine angle. Paralysis of the seventh nerve may occur on one side with a tumor in the angle of the other side, because the tumor pushes the cerebellum over to the opposite side, compressing the facial nerve of that side against the petrous portion of the temporal bone.

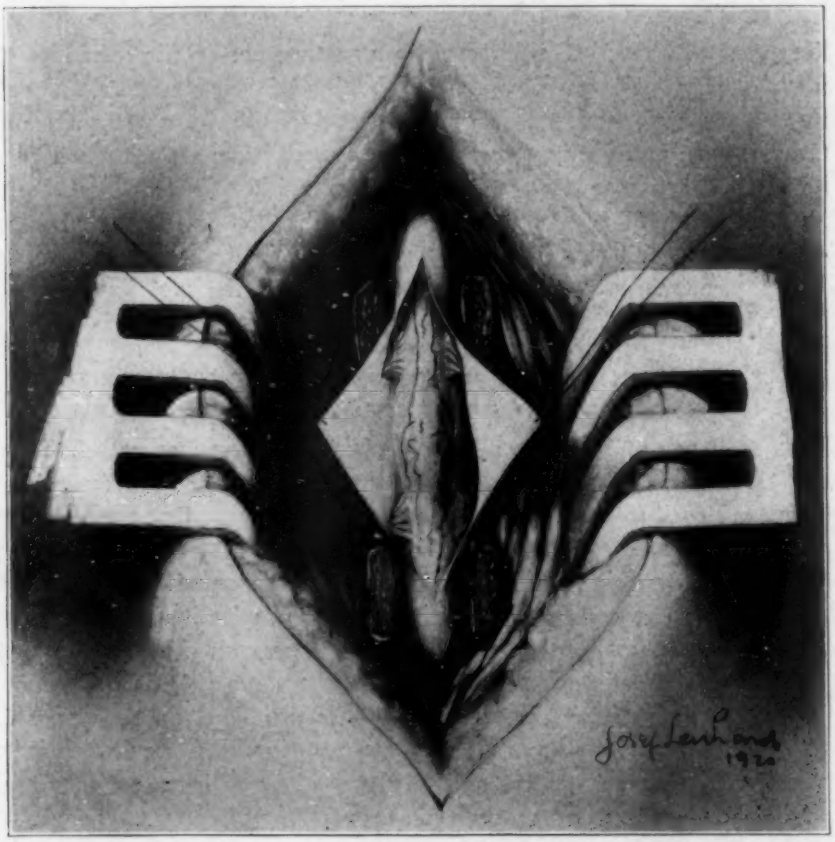

Fig. 9.—Intramedullary tumor in the posterior column. The growth is covered by a thin layer of cord tissue.

Especially in patients who give no history of root pains, the occurrence of more marked motor symptoms (and also, sometimes, of more distinct posterior column disturbances) on one side may mislead the examiner, so that the growth is localized on the wrong side of the cord. These cases are examples of reverse Brown Séquard symptoms and may occur when a tumor of soft consistency pushes a movable part of the cord to the opposite side.

It is possible also that in some patients with bilateral sensory and motor symptoms, the signs of interference with the tracts on the side of the tumor are due to direct pressure by the growth, and those on the other side are caused by the pressure of that side of the cord against the wall of the spinal canal. Such a mechanical condition would result in the kind of double Brown Séquard syndrome that has been described by some authors.

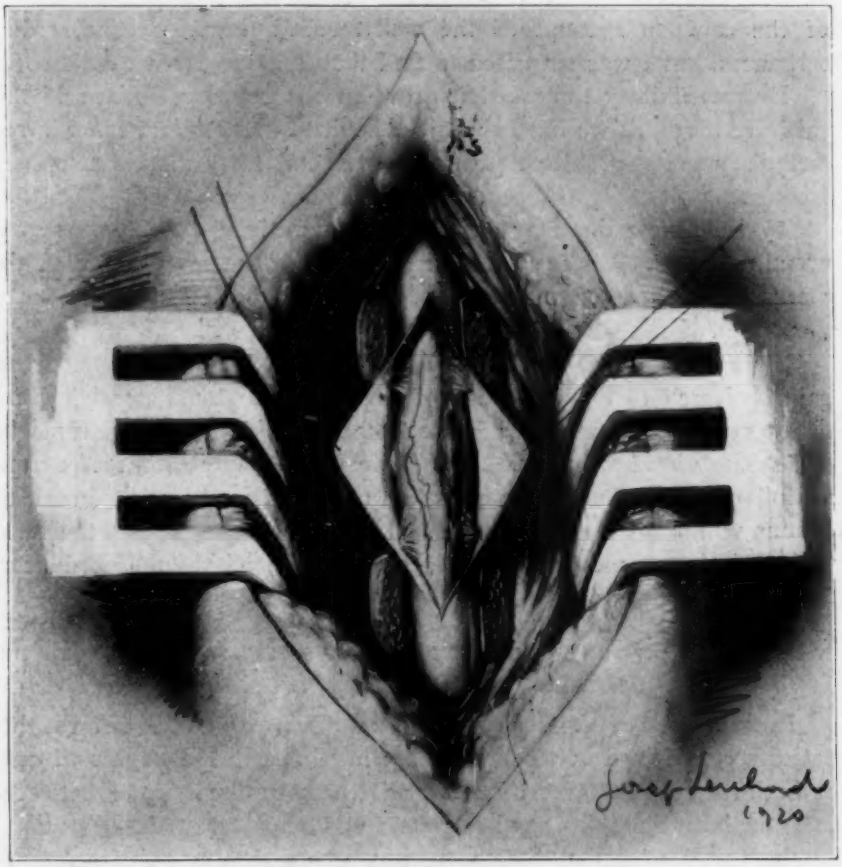

Fig. 10.—Partial extrusion of intramedullary tumor after an incision into the cord had been made.

As is well known, the fibers that convey the so-called deep sensations, the sense of position, of movement and of weight, the vibratory sense, and tactile discrimination, travel up the spinal cord in the posterior column of the same side. When these sensations are disturbed on only one side of the body, the diagnosis of a lesion on that side of the cord can be made with a fair degree of certainty. In many patients there is evidence of disturbance of both posterior

columns although one side is affected more than the other. In some patients, however, the signs of interference with both posterior columns are the same on both sides, and in such a case the question may arise whether the new growth lies on the posterior surface of the cord and is compressing both posterior columns, or whether the tumor is growing in front of the cord and is pushing the cord backward against the arches of the vertebrae. I have seen one case of this kind. In this patient I expected to find the tumor on the posterior surface of the cord on account of the well marked posterior column disturbances, but I was surprised to find that it lay in front of the cord.

Extramedullary tumors that grow on the anterior surface of the cord are often, as we have shown, difficult to differentiate from intramedullary growths. Root pains are rare and early muscle atrophies frequent.

Typical root pains may occur with intramedullary tumors, as may even unilateral posterior column disturbances, if the new growth within the cord substance is small and located in one posterior column near the origin of a posterior root.

Figures 9 and 10 illustrate this condition. The patient had root pains referred to the right side of the thorax and unilateral loss of vibratory and articular sense on the same side, with a spastic paraplegia and sensory level symptoms. The history was a typical one of extramedullary tumor, but at the operation a growth in the right posterior column within the cord was found. There is much to support the view that typical root pains can follow an affection within the cord near the origin of a posterior root.

PARESTHESIA IN ONE OR BOTH UPPER EXTREMITIES CAUSED BY TUMORS IN THE THORACIC CORD

In five cases of thoracic cord lesions of an expanding nature, the patients complained of tingling or hyperesthesia in the fingers of one or both hands. In three of these, an extramedullary tumor between the fourth and eighth dorsal segments was removed at the operation. In the first two patients we were much confused by this symptom, because it did not agree with the other level symptoms. In one of the patients, in fact, the spinal symptoms were supposed to be due to a diffuse lesion and the surgical interference was delayed for a considerable period on account of the symptoms in the upper limbs. One patient had been walking around on crutches, and we believed the tingling in the forearm and hands was due to pressure on the nerves in the axilla by the crutches. The other patients were bedridden, and in their cases some other explanation had to be found. I believe that the tingling in the fingers in these cases was due to a slight pres-

sure on, and irritation of, posterior roots by a column of cerebrospinal fluid above the tumor. In at least one of the patients, a large amount of fluid under great pressure escaped from above as soon as the tumor was removed. In all of the patients, the cutaneous hyperesthesia and the tingling had disappeared when the patient recovered from the anesthesia.

Abstracts from Current Literature

UEBER MYOKYMIE UND MUSKELVERAENDERUNGEN BEI SKLERODERMIE (CONCERNING MYOKYMIA AND MUSCLE CHANGES IN SCLERODERMA). S. NEUMARK, Schweizer Arch. f. Neurol. u. Psychiat. **6**:125, 1920.

The syndrome of myokymia was first described by Kny (1888) and Morvan (1890), the latter designating the condition as chorée fibrillaire. Schultz, in 1895, gave it the name it now bears. The cardinal symptom consists in localized, wavelike muscular contractions without motor effect. While there is a symptomatic myokymia which occurs in a large variety of organic diseases of the nervous system, the so-called essential type develops in persons who have previously enjoyed good health, but who have, in most instances, been subject to a slight infection; the condition disappears spontaneously.

Neumark reports a case presenting some unusual features. The patient was a man, 65 years of age, who had been seen the first time fifteen years previously, when a diagnosis of psychasthenia was made. Even then it was noted that the facies was mask-like; there was a constant wrinkling of the brow and fascicular contractions spreading over the entire face gave one the impression of slow, grimacing movements. The patient could bring these movements to rest, at will, for a short time, when the mask-like appearance became striking. A Chvostek II was noted; however, Trousseau's sign was absent; subsequently the patient developed attacks of unconsciousness. At the time of his examination, there was a tremor of the tongue and a fibrillary twitching along the edges, but no atrophy. Fascicular twitching was present over the entire face, but more marked on the left than on the right. There was still a continual wrinkling movement of the brow, which was aggravated by fatigue and by emotional disturbances. Aside from a sensation of tension there was no subjective sensory disturbance. In addition to the fibrillary tremor, there was a left-sided facial spasm. The mandible was somewhat atrophic; the scalp was thin, smooth, glistening and adherent; over the left mandible, a sclerodermic plaque was situated; and in addition, sclerodermic changes involved the hands and possibly the thigh. The hands were blue and cold and the lower extremities showed a marked cutis marmorata. There was a slight dorsal kyphosis. No sensory disturbances could be demonstrated objectively. The writer laid great stress on the fact that the movements about the face ceased entirely during sleep. Atrophic changes of the bones of the hands could be demonstrated by the roentgen ray. Aside from these symptoms, the examination was practically negative.

Neumark, in reviewing the reported cases of myokymia, found that the seat of the disturbance commonly involved the lower extremities, particularly the calves, although almost any muscle of the body might be involved. The etiologic factors given were overexertion, coryza, trauma, chronic lead poisoning, syphilis, tuberculosis, chlorosis and the neuropathic habitus. Men were principally affected; age ranged from 11 to 65, and no type of occupa-

tion was excepted. Muscular irritability was increased in most cases, as was also the excitability of the nerve trunks. The reflexes were usually increased. Vasomotor disturbances were common. The duration of the disease varied; in the majority of instances the movements disappeared in a few weeks, although they have been known to persist for twenty years, and in the case reported they had been present for more than ten years. The writer called attention to the fact that, so far as he is aware, no case of a myokymia complicated by scleroderma, has been described; various types of muscular atrophy associated with scleroderma are by no means uncommon. The part in the nervous system from which these movements arise is not known, although there is reason to believe that it may be situated in any part of the reflex-arc or even in the muscles themselves, as Oppenheim supposes. Kurschmann attributes the movements to neuritis. It is believed that there is a close relationship between myokymia and myotonia acquisita of Talma. Neumark thinks that the so-called essential myokymia is not dependent on an organic lesion of the nervous system, but is the expression of a functional disturbance.

In discussing scleroderma, the author quotes Hutchinson to the effect that associated muscular atrophy is the rule and is absent only in mild cases and in old people. The writer's patient was well along in years, which may explain the absence of muscular atrophy. The types of muscular disturbances noted in scleroderma may be classified as atrophy, myosclerosis and myositis, although none of these commonly occur alone; as a rule, there has been found a primary increase in the interstitial connective tissue with a resulting pressure atrophy of the muscle fibers, as well as a myositis. Insufficiency of the functions of the skin, compression of the muscles, blood vessels and nerves all contribute to the atrophy. Cases have been reported, however, in which the muscular atrophy preceded the cutaneous changes by several years and progressed independently of changes in the latter. Disturbances in the function of the skin, such as profuse perspiration, may also precede. Furthermore, myosclerosis may occur in a location of the body quite distant from the changes taking place in the skin. Myositis ossificans has been seen in association. It has been assumed that the muscular change of scleroderma may be the result of a general infection-intoxication process which begins as a myositis and ends in sclerosis. The involvement of the skin in this inflammatory process as well has been assumed and the entire process looked on as a dermatomyositis. Cassirer takes this standpoint for some of the cases and it is the one to which the writer adheres. A differential diagnosis between these and the other types of scleroderma may therefore be impossible. The histopathologic changes noted show an atrophy of the muscle parenchyma, peri-arteritis and endarteritis, endophlebitis, perimuscular and intramuscular sclerosis, disappearance of the cross-striations, splitting of the muscle fibers, homogenization of the protoplasm, and small round cell infiltration. While the reports of different investigators differ in some respects, they all seem to emphasize the importance of vascular changes in bringing about the disintegration of muscle. It is true that the former may in turn be due to some lesion of the nervous system. The writer is unable to say what relation, if any, the myokymia in his case bore to the scleroderma; he is inclined to think, however, that they are two separate and distinct entities, although they may both be manifestations of one and the same general neurosis.

WOLTMAN, Rochester, Minn.

UEBER DIE PSYCOPATHISCHE KONSTITUTION BEI KRIEGS-NEUROSEN (THE PSYCHOPATHIC CONSTITUTION IN RELATION TO WAR NEUROSES). FRITZ FRÄNKEL, Monatschr. f. Psychiat. u. Neurol. **47**:287 (May) 1920.

The majority of investigators believe that a constitutional predisposition is an important factor in the development of war neuroses. The author analyzes seventy-two cases of war neurosis with this point in view.

The concept of "psycopath" lacks any exact objective criteria. The stigmas of degeneration are valueless, and our judgment must be based on the past history of the patient. The demarcation between normal and psycopath is gradual and, in a sense, arbitrary. Arbitrary also is the grouping of the psycopathic states, for there are all grades and all transitions. The author adopts the following classification: imbeciles, 8; unstable (haltlose), 5; unbalanced (verschobene), 3; constitutionally irritable, 14; constitutional neurasthenics, 10; sensitive, 19, and cyclothymic and depressive, 2. The different classes are discussed at some length.

The unstable (haltose) are characterized by the instability of their lives. One of this group held twenty-four positions in a short time. Many of this group, 54 per cent. according to Kraeplin's statistics, come into conflict with the law. This class includes many pathologic swindlers and pathologic liars.

The unbalanced (verschobene) are characterized by a sense of superiority and a heightened sense of self regard. They feel superior to their station in life and are always reaching out for higher levels both socially and intellectually. They are unstable in their relations to the opposite sex.

The constitutionally irritable (konstitutionel erregte) form the second largest group. They lack the normal inhibitions and self restraint, fly off the handle, fight at the drop of the hat and are given to outbursts of violence. In civil life these people have a chance to relieve the accumulating tension by emotional outbursts, but the strict army discipline prevents this and causes increased emotional repression, so that they seek the only way out—escape in sickness. In no class of psycopath is the character of the war neurosis as defense reaction more evident than in this class. It includes the most primitive reaction types whose protest character is grossly demonstrated in a paroxysm of striking, kicking and shrieking or in blocking of consciousness in unconscious spells or dream states.

Characteristic of constitutional neurasthenics are hypochondriacal complaints of all types. They suffer from every conceivable sort of bodily weakness. Objectively, we find vasomotor instability. It is uncommon for this group to stand any long period of field service.

The sensitive group includes timid, dependent persons, abnormally sensitive to any threat of physical violence. They resemble the neurasthenics in the tendency to vasomotor instability. Their psycopathic trend shows itself in early childhood. They cannot see any one hurt, cannot endure see-saws and merry-go-rounds, and are subject to night terrors and anxiety dreams. Later, as adults, they are timid in the presence of strangers and easily embarrassed. They are seclusive and have few friends. They are shy in the presence of women, and apt to be sexually abstinent until marriage. Alcohol intolerance is common. This type includes the largest number of cases.

Summarizing, the author states that the endogenous factor—the psycopathic constitution—is most important in determining the causation of war neuroses. Exogenous factors are much less significant. Sixty-one of the seventy-two cases show this clearly in the past history. Of the eleven others, whose his-

tory was not so definite, there was not one who presented a normal personality. According to Gaupp, even if the family history shows no evidence of mental or nervous disorder, and the patient states that he has always been well, the mere fact that he developed symptoms in a situation which left his comrades unaffected, demonstrates that the essential cause of his trouble lies within himself. The objection may be raised that this makes the concept of the psycopathic constitution too broad. But the fact is that the great experiment of the war proved that the number of abnormal personalities was vastly greater than we formerly believed. The war brought them out. There is danger in this, because the laity has come to feel that groups of nervous symptoms are the natural result of mechanical and psychic traumas. We may have a mass development of such states as traumatic neuroses. It behooves us as physicians to educate people in the belief that we are capable of standing even exceptional traumas without developing symptoms, and that when symptoms do develop under strain they result from our own weakness rather than from the trauma itself.

SELLING, Portland, Ore.

RUPTURE OF THE SPINAL CORD IN DYSTOCIA. F. H. KOOY, J. Nerv. & Ment. Dis. **52**:1 (July) 1920.

The patient whose case is reported was the fifth child. The first two children were in transverse position when labor began; the third and fourth were in normal position. The patient was also in transverse position in utero, with the head offering some difficulties, and was revived with difficulty after birth. Three days later, ulcers appeared on the buttock which rapidly increased in size. The child could never move its lower legs, feet or toes; there was only slight flexion at the hip. At 1½ years, the child burned its legs without pain. It had always had incontinence of urine and feces. At the age of 2 the legs were paretic and spastic, with slight movement at the hip. Thermic sense was lost in the legs and gluteal region: there was a large, deep bedsore on the sacrum. While in the hospital, he suffered a spontaneous fracture of the femur. At the age of 7, the paralysis became atonic with loss of Babinski sign, with knee jerks diminished and ankle jerks absent. Touch was lost in the lower part of the legs and diminished in the upper part of the legs. An abscess over Poupart's ligament was incised without pain; the decubitus was increased in size; incontinence of urine and feces continued. At the age of 8, knee jerks were lost. The patient died at the age of 9. The diagnosis was lesion of the cord resulting from dystocia.

Postmortem examination demonstrated that the decubitus had penetrated the sacrum; there was no abnormality of the vertebral column. At about the seventh thoracic vertebra the cord was found to be a thick, solid ring of fibrous tissue with adherence of the dura. Practically the entire cord was examined in serial sections, alternately stained with Van Gieson's and Weigert-Pal's methods. At the level of the lesion the structure of the cord was unrecognizable. There was a network of connective tissue with small islands of glia and a small bundle of nerve fibers which when traced down proved to be the direct pyramidal tract. On this finding the author explains the small amount of movement at the hip. The ascending and descending degenerations of the cord were very painstakingly worked out. Marchi's stain, of course, could give no information since the case was of too long standing but with the Weigert method the author traced the various tracts.

He classifies lesions of the spinal cord dependent on dystocia as occurring in three different ways: (1) by venous engorgement, compared to hemorrhages in the adult in states of sudden venous congestion from vigorous movements, etc. He states that published reports of cases in which venous congestion is probably the only cause of these hemorrhages in the spinal cord of the newborn are not many, and that multiple hemorrhages are commonly found; (2) by fracture, rupture, or luxation of the vertebral column and a secondary meningeal and medullary hemorrhage with more or less complete compression of the cord; (3) partial or complete rupture of the cord, with the vertebral column intact. But he also adds that it is impossible to tell by microscopic examination at all times the precise mechanism of its origin, and most difficult of all is the differentiation of a rupture of the cord from hemorrhage, as a large hemorrhage may cause a complete destruction of the spinal cord.

The author discusses the question of the reflexes (Bastian's law) but does not quote any of the modern war literature on the subject. The cause of the patient's decubitus is rather interesting since it appeared the first few days after birth and pressure could not have been responsible. The author decides in favor of trophic influences. He also discusses the fact that although the child could flex both hips, only one anterior pyramidal tract was intact, and he reaches the conclusion that each ventral pyramidal tract serves as a path for bilateral innervation of the legs.

WINKELMAN, Philadelphia.

THE RESULTS OF SECONDARY SUTURE OF PERIPHERAL NERVES.

JOHN S. B. STOPFORD, Brain **43**:1 (May) 1920.

In this article are reported the preliminary results of secondary suture of nerves in 271 cases. The author systematically discusses the general factors which influence the progress and results, the technic of repair, conditions found at operation, complications and the comparative results of suture of the different nerves.

The delays to operations that are encountered after primary wounds are mainly sepsis, and in four cases ununited fractures. A period of from twelve to eighteen months' delay in suturing does not necessarily offer an obstacle to regeneration; but sutures in proximal parts offer decidedly better prognosis, and the larger the nerve trunks, the greater the amount of regeneration. The best operative technic is freeing the nerve ends, with wide resection to normal nerve tissue, suture without torsion through the neural sheath and with one through-and-through tension suture. Embedding the nerve in a new place, preferably in healthy muscle, insures against secondary scar formation and subsequent compression. Wide resection of ends in incomplete divisions is indicated to gain the maximal regeneration, as it has been shown that greater contractures and mechanical disabilities are associated with incomplete divisions.

In the presence of sepsis, intraneural changes take place extensively, even so much as 8 inches above the point of injury. Ligature of the main artery in the proximal part of the limb is mentioned as an aid to restoration, only to be condemned. In ununited fractures, the bone condition should be repaired first and after this has healed, the nerve can be sutured with much better chances of success.

As to results in secondary suture, none are perfect; there is always a deficit, and in this series, sensory regenerations were particularly disappointing. As to factors which hinder complete recovery: Intraneural changes in the proximal end, obstruction to the downgrowth of new nerve fibrils, destruction

of nerve branches and "bad shunting" are cited. Further, the loss of afferent stimuli from joints, muscles and tendons accounts for the disability of the limb, for whereas the individual muscles show return of power and the superficial sensation is fairly completely restored, yet the patient is unable to use the limb well except when he is watching it, and has lost those vital necessities for definitive use—the sense of position and appreciation of movement.

Suture of the musculospiral nerve gave uniformly good results, particularly when the injury was high up. The nearer to the cord, the more complete and rapid the return of function. Loss of synergic action in the extensors of the muscles accounts for the weak hand grasp and poor recovery that are generally encountered in the thumb extensors.

In the median nerve suture, an incomplete recovery of protopathic sensibility was general, and recovery of the voluntary power in the abductor brevis pollicis occurred in but 40 per cent.

The ulnar nerve recoveries were disappointing, possibly from "bad shunting" and from the small nerve supply to the intrinsic muscles of the hand, rendering the chances greater of their not receiving efferent fibers. Brouwer's hypothesis that the vulnerability of these finer muscles and nerves is due to the "finer construction of the thumb and higher functional significance which it has received in the phylogenesis" is offered as a suggestion in explanation.

In the sciatic, return of function in the tibialis posticus and flexors of the toes was poor. The external popliteal failures are accounted for by the inability to procure a good bed after suture, and the tendency to involvement of the nerve in scar tissue. In the internal popliteal, functional results obtained were good except in the restoration of sensation. End-to-end suture was done in all cases, and no nerve grafting or splitting operations were performed.

PATTEN, Philadelphia.

VISUAL FIELD FINDINGS IN A CASE OF BRAIN TUMOR. WALTER R. PARKER, Am. J. Ophth. 3:736 (Oct.) 1920.

J. M., aged 28, first seen in 1911, a sailor, was sent home on account of inattention and inability to keep awake. Four years before he suffered attacks of headaches. About two years later headache increased and was often accompanied by nausea and vomiting, with occasional brief attacks of vertigo and falling sensation. He never complained of loss of vision. In 1911 vision was: right eye, 6/15; left, 6/12. Pupil reaction was present but was somewhat sluggish, especially in the right eye. Wernicke's sign was present. There was edema of both disks, most marked in the right eye, but there was no measurable swelling.

The headaches did not increase in severity, but mental and motor disturbance gradually became more marked. When he developed symptoms of hyperpituitarism a sellar decompression operation (transsphenoidal route) was performed by Dr. R. B. Canfield. Shortly after this symptoms of hypopituitarism appeared.

The patient died April, 1914. Postmortem examination showed a tumor involving the right tract, chiasm and adjacent brain substance. Pathologic Report: Large round celled sarcoma with numerous calcareous concretions (psammoma); growth probably primary in the meninges.

The visual fields showed first a homonymous hemianopsia without involvement of the muscular region. Vision in the right eye was 6/15; in the left eye, 6/12. Later the macula became involved and vision in the right eye was

1/60; in the left eye, 5/30. Still later there was a loss of green perception in the right eye with a marked contraction of the temporal form field. This was followed by loss of all color sense in the right eye with marked temporal contraction for form. Vision: Fingers could be seen at a distance of 2 feet. Finally there was total blindness in the right eye.

Both optic disks gradually paled and became atrophic. Examination by Dr. C. D. Camp, March 11, 1912, revealed general muscular weakness, slight difficulty in turning to the right, no nystagmus, power of convergence normal, slight facial paralysis, a positive Romberg sign, the knee reflex markedly exaggerated and a slight memory defect. The Wassermann and urine tests were negative.

Discussion.—Apparently the tumor first involved the right tract inducing a left homonymous hemianopsia and a positive Wernicke's sign. Later it either invaded the chiasm or led to pressure symptoms that affected only the papillomacular bundle causing loss of central vision. Finally a possible bitemporal hemianopsia developed, and this superimposed on a homonymous hemianopsia led to blindness in one eye (right) and loss of the temporal field in the other.

REESE, Philadelphia.

EPIDEMIC ENCEPHALITIS: INCLUDING A REVIEW OF ONE HUNDRED AND FIFTEEN AMERICAN CASES. ARTHUR D. DUNN and FRANCIS W. HEAGEY, Am. J. M. Sc. **160**:568 (Oct.) 1920.

The authors have carefully analyzed 115 cases of epidemic encephalitis. The frequent incidence of respiratory infections (31 per cent. occurring in close relationship to the development of encephalitic symptoms) is a point of etiologic interest. The various forms described, polio-encephalitic, lethargic, parkinsonian, cataleptic, meningitic, cerebral, polyneuritic and myelitic, must not be regarded as clear-cut clinical types. Not only is there considerable overlapping, but the symptomatic complexion of the individual case frequently changes during the course of the disease. Lethargy was noted in 79 instances, third nerve palsies in 66, diplopia in 58, headache in 57, fever in 56 and sixth nerve palsies in forty. The occurrence of catalepsy and catatonia in twenty-six cases is in line with the experience of other observers, and is an addition to the literature on this subject. In the face of the incidence of catatonia in the greatest diversity of conditions, not only in the psychoses and psychoneuroses, but also in organic brain diseases and infectious diseases, it is difficult to understand why it is still so often regarded as synonymous with dementia praecox and further why it is often given a purely psychologic interpretation.

The serologic analyses demonstrated that in twenty-five counts on fifteen cases there was an average leukocytosis of 10,200 with a neutrophilic percentage of 71; in sixty-four spinal fluid examinations there was an average of sixteen cells per c.mm., chiefly of the mononuclear variety with a positive globulin test in 50 per cent. of the cases and a mild syphilitic gold chlorid reaction in seven of eleven fluids. At present we are restricted to the following pathologic picture: (1) meningeal edema and thickening; (2) softening and congestion of both gray and white matter of the brain and pituitary gland; (3) punctate hemorrhage in mesencephalon and thalamus and basal ganglions; (4) thrombosis of small vessels; (5) perivascular infiltration of small vessels of the brain stem; (6) edema of the mesencephalic area.

The meager extent of our etiologic knowledge is reflected in the far from favorable prognosis. In 100 cases reported in the American literature, thirty-one patients died. The treatment is necessarily in the symptomatic stage. Lumbar puncture is of decided benefit.

STRECKER, Philadelphia.

FORMS OF PERIPHERAL NEURITIS AMONG TROOPS SERVING WITH THE EGYPTIAN EXPEDITIONARY FORCE, 1915-1919. F. M. R. WALSHE, Brain **43**:74 (May) 1920.

The commonest organic nervous diseases encountered in this campaign were multiple neuritis and the postdiphtheritic paralyses. No cases were seen due to malaria or dysentery, nor were there any instances of acute febrile polyneuritis. The total number was 160, one group of which followed faucial diphtheria, the other followed diphtheritic infection of skin wounds.

Of the faucial type, patients developed palatal paralysis in the second or third weeks of illness. Diminution or abolition of knee reflexes were not initial symptoms. Subjectively, aching pains in the legs on exertion, painful cramps in muscles, numbness of the feet, tenderness of muscles, and lively tendon jerks were the premonitory manifestations. The tendon jerks, however, usually diminished later.

In the 1916-1917 epidemic of diphtheria, the neurologic involvement reached a high percentage because of small dosages of antitoxin, and the premature return of patients to physical activity.

Sixty cases of multiple neuritis followed septic skin "sores," in several of which the diphtheria bacillus was isolated. The relation of the condition to diphtheria was practically certain; the patients came from areas where diphtheria was prevalent, and all gave histories of sores on the exposed skin surfaces which yielded slowly to treatment. The appearance of signs and symptoms in these cases followed a fairly definite order. There was first local paresis in the region of the site of the infective focus; then two to three weeks later paralysis of accommodation, and still later polyneuritis. Loss of accommodation was present in but one third of the cases; but infective foci, multiple usually, and polyneuritis were common to them all.

The author believes, in the light of the anesthesia and paralysis about the focus of skin infection, that the palatal paralysis in faucial diphtheria is a true local palsy. Consequently, despite the location of diphtheritic infection, the symptoms are clinically the same. This leads to the hypothesis that the toxins are conveyed by the perineural lymph channels, and therefore the nerves nearest the focus of infection are the first to become paralyzed through the involvement of those segments in the central system. Selective action is pointed out as a probable explanation of the paralysis of accommodation and a blood-borne toxemia causing the polyneuritis.

Of interest is the resemblance of this condition, following the above hypothesis, to tetanus.

PATTEN, Philadelphia.

TRAUMATIC REFLEX IMMOBILITY OF THE PUPIL. B. FLEISCHER and E. NIESENHOLD, Klin. Med. f. Augenh. **64**:109 (Jan.) 1920.

After a review of the literature, a case is described in an otherwise healthy girl, 17 years of age, seen six days after her left lower lid was struck by a hay fork. The oculomotor nerve was completely paralyzed; the pupil was wide without reaction; abducens and trochlearis were functioning; the disk was not sharply defined; the surrounding retina was opaque; vision was 2/12.

The other eye was normal. The condition improved to a certain extent. After thirteen months the temporal portion of the disk was pale; vision, 1. The field was normal; there was some diplopia.

There was no reflex immobility, but an incomplete absolute unilateral immobility with almost complete abolition of direct and diminished consensual reaction to light; there were impaired accommodation and partial persistent paralysis of exterior muscles, caused by an orbital injury, similar to Laquer's case. The consensual, although incomplete, reaction with almost entirely lacking direct reaction, is attributed to the partial optic atrophy, inhibiting the conveyance of the lighter stimulus, and not able to arouse the function in the damaged light reaction fibers of the oculomotor nerve. The prompt consensual reaction of the other pupil proved that the transmission of the light stimulus in the optic nerve was not suspended.

The authors are tempted to assume in this case that a special damage to the pupillary fibers of the optic nerve occurred. They believe that also in the other traumatic cases the seat of the disturbance must be sought in the periphery, mostly in the orbit. On account of the almost missing direct, and insufficient consensual, reactions the authors assume a rather feeble impulse of convergence, through the existence of special convergence fibers, which escaped the injury, damaging only the light reaction fibers. The lesion might have damaged the ciliary ganglion, so that the finer transfer of the stimulus in the ganglion was rendered difficult or impossible; but the fibers penetrating or passing by the ganglion, namely, the supposed convergence fibers, were not affected.

REESE, Philadelphia.

THE DEVELOPMENT OF THE SYMPATHETIC NERVOUS SYSTEM IN MAN. ALBERT KUNTZ, J. Comp. Neurol. **32**:173 (Oct. 15) 1920.

In a series of about ten publications, Kuntz has reviewed the development of the sympathetic nervous system in representatives of the leading groups of vertebrates, finding in all cases certain broad similarities. The primordia of the sympathetic trunks and the prevertebral plexuses arise from cells of cerebrospinal origin which advance peripherally along both the dorsal and ventral roots of the spinal nerves. The vagal sympathetic plexuses, namely, the pulmonary, the cardiac and the enteric plexuses, except in the aboral portions of the digestive tube, arise from cells of cerebrospinal origin which advance peripherally along the vagi. In the more distal portions of the digestive tube the enteric plexuses arise from cells which are derived from the sympathetic supply in the lower trunk region. The ganglions of the head sympathetic of mammals were studied chiefly on the pig.

These results have been in part confirmed and in part adversely criticized by later students of the subject, the account of the origin of the cranial sympathetic ganglions having been especially attacked by Stewart (*J. Comp. Neurol.* **31**: 1920), who studied the rat.

Kuntz has now restudied the entire question on the basis of an examination of the extensive series of human embryos in the Mall collection. Regarding the controversial points in the development of the cranial sympathetic ganglions, he confirms most of his earlier observations made on the pig and at the same time admits the correctness of Stewart's findings. This implies that most of the ganglions of the head sympathetic nerves have a double origin, only one component of which was recognized in the author's earlier studies. For the details we must refer to the original text, which is accompanied by numerous photographs of the preparations.

HERRICK, Chicago.

ZUR HETEROTOPIE DER PLEXUS CHOROIDEI (CONCERNING HETEROTOPIA OF THE CHOROID PLEXUSES). S. KITABAYASHI, Schweizer Arch. f. Neurol. u. Psychiat. **6**:154, 1920.

In the course of Kitabayashi's study of the choroid plexus in schizophrenia, he discovered an interesting heterotopia of the cerebellum and of the choroid plexuses, which up to the present time had not been described. Heterotopias and heterotaxias of the cerebellum are not rare. The one which the author describes differs in some respects from those heretofore reported. The patient from whom this particular specimen was obtained was a male schizophrenic (catatonic), 29 years of age, who died of tuberculosis after an illness of five years.

Three distinct heterotopias were noted. The first of these was located in the region of the tuberculum acusticum, the condition being seen best in the section cutting this structure, the corpus restiforme and the velum medullare inferius. The wedge-shaped projection of the flocculus contained heterotopic portions of choroid plexus embedded partly in the granular layer and partly in the medullary substance. No ependymal cells were included with the heterotopic villi.

The second anomaly was located in the oral portion of the horn of Ammon near the fimbria, and consisted of two abnormal cavities, in the form of a figure eight, filled by irregular masses of plexus villi. These spaces also were not lined with ependymal cells.

The third was located in the medullary substance, midway between the corpus geniculatum externum and the nucleus caudatus, about the location of the taenia semicircularis, where a small cavity was seen, which was connected with the ventricle and into which the villi of the choroid plexus projected filling it entirely. This cavity was lined with ependymal cells. Microscopic examination showed the heterotopic villi to be practically normal in structure. The third heterotopia, it was believed, might have arisen in a late embryonic stage or might have been acquired even later; the first two, however, must have developed at an early embryonic period on the basis of misplaced cells of the choroid plexus.

WOLTMAN, Rochester, Minn.

HEMORRHAGE INTO THE SPINAL CORD AT BIRTH. CHARLES W. BURR, Am. J. Dis. Child. **19**:6 (June) 1920.

The first case reported is that of a boy, 4½ months old, who had four convulsions in the twelve hours prior to admission to the hospital. The previous history disclosed the fact that the birth had been a breech presentation, and the right shoulder had been hurt in delivery. The symptoms had apparently been present since birth, but had been overlooked. Examination demonstrated the position to be typical of that of a transverse cervical cord lesion; there was palsy of the external muscles of respiration with flaccid paralysis of both legs, with reflexes of defense, and anesthesia of the legs and trunk. A roentgenogram of the spine was negative. Lumbar puncture produced a fluid at first clear but tinged with blood at the end, containing ten cells. The Wassermann test was negative. The child died of pneumonia. At necropsy no signs of vertebral injury nor signs of old extradural hemorrhage were found. The cord and membranes from the fourth cervical to the first dorsal were a soft fibrous band. No hemorrhages were found in the viscera. Microscopic examination by Dr. A. J. Smith demonstrated on section of the involved area

that gray matter was not recognizable. There were no multipolar cells and only a few patches of degenerated white matter with no normal white matter so that at this level the cord was practically destroyed. There was ascending and descending degeneration above and below the site of the lesion.

The second case is similar as to history and physical findings, but no necropsy was obtained.

The author concludes from the necropsy findings of the first case that there was a hemorrhage into the cord and a subsequent myelitis, and he is inclined to the belief that the hemorrhage was spontaneous and was not caused by direct violence but by rupture of overfull vessels from pressure on the soft parts during birth.

WINKELMAN, Philadelphia.

UN CAS DE PARALYSIE SYPHILITIQUE DU NERF CUBITAL A LA PERIODE TERTIARE (SYPHILITIC PARALYSIS OF THE ULNAR NERVE DURING THE TERTIARY PERIOD). PIERRE ROBLIN, Bull. méd. **34**:838 (Sept. 18) 1920.

The writer has found only four records of syphilitic paralysis of the ulnar nerve in the literature. Gaucher has described the condition and considers it a secondary stage manifestation. In the case which Roblin reports, the condition was clearly a tertiary stage development.

A man, aged 38, without any previous knowledge of syphilitic infection, was seen by the writer in 1909, because of a slowly enlarging, painless, hard tumefaction of the lower lip. It was pronounced syphilitic, and under mercurial treatment disappeared in about forty days. Treatment with considerable vigor was then continued for two years.

Approximately six months later, there developed a right ulnar neuritis with paralysis. The features, typical of an ulnar (incomplete) lesion, need not be enumerated. They included pain on pressure over the hypothenar eminence. It was established that the lesion was not due to a bony exostosis which had "caught" the nerve. There was a dilatation of the left pupil which, however, reacted well to light. The treatment included potassium iodid and injections of mercuric benzoate in two series; and locally, massage and electricity. This led to a rapid amelioration, and the affected movements became almost normal in two months and completely so after five months. At the time, the inequality of the pupils remained.

The writer asks whether the pupillary change compels one to diagnose a central nervous system lesion, or whether the ulnar lesion was actually peripheral as appeared to be the case.

DAVIS, New York.

THE GERMAN INSTITUTE OF PSYCHIATRIC RESEARCH. EMIL KRAEPELIN, J. Nerv. & Ment. Dis. **51**:6 (June) 1920.

Kraepelin, in this article, stresses the need in Germany of extension of the scope of work done at the Institute for Psychiatric Research at Munich. Inquiry is needed into the many prenatal causes of disease with particular emphasis on the rôles played by alcohol, syphilis, infections and injuries, as well as the inheritable deficiencies which have heretofore received little attention. There is a broad field for investigation in the line of preventive medicine and biochemistry. It is stated that the investigations must necessarily reach out into every phase of the physical and mental life of the people: the

economic, developmental, religious, educational, artistic, literary and national life. Incidence of birth rate and control of death rate, marriage, and age of mortality should be sought out through statistical research in order that there might be constructed "a general picture of the folk soul." The research should aim to "make clear the nature and the sources of mental disturbances" and then "discover ways of preventing them, healing them, or making them easier to bear."

The furtherance of such work necessitates adequate appropriation for the endowment of positions and the work must be so arranged that younger men would be attracted to the field, who would be able to give their entire time to it, free from all other conditions which might interfere, such as earning a livelihood by other means. To the existing departments in serology, anatomy, and demographic-genealogical research, it is proposed to add departments in chemistry and psychology.

The author strikes the keynote in psychiatric research when he emphasizes the demands for inquiry into the underlying factors and predisposing causes of disease. Preventive medicine, although well recognized, has many limitations thus far, and greater cooperation and added facilities are requisite to make it more efficacious. This condition can be brought about through appropriations from the state and the enactment of necessary laws to enforce preventive measures. The outline of the work and suggestions given in this paper undoubtedly echo the thoughts and desires of many American psychiatrists, who are still struggling to place research in mental disease problems on a better scientific basis.

PATTEN, Philadelphia.

ANGINE DE POITRINE GUERIE PAR LA RESECTION DU SYMPATHIQUE CERVICO-THORACIQUE (ANGINA PECTORIS CURED BY RESECTION OF THE CERVICAL SYMPATHETIC NERVE). T. JENNESCO, Bull. de l'Acad. de méd. **84**:93 (Oct. 5) 1920.

The symptoms of angina are caused by irritation of the cardio-aortic plexus by a lesion in the aortic wall. The reflex which arises from this lesion brings about vascular, painful and motor disturbances which constitute the syndrome known as angina. These disturbances can occur only when the nervous reflex originating in the aorta can reach mesencephalo-medullary centers.

By destroying the centripetal pathway for this reflex through the resection of the cervical sympathetic nerve (left), the anginal attacks are no longer possible.

Full data is given of a man aged 32, a syphilitic, with a specific aortitis and a history of severe frequent anginal symptoms, not benefited by mercurial treatment. The removal of the left cervical sympathetic nerve relieved him abruptly of these attacks. He was seen four years after the operation, and relief had continued during that period.

The writer believes that sudden death, so frequent in angina, is due to bulbar anemia provoked by spasm of the arteries; this is in turn a reflex from aortic irritation. In that event resection of the cervical sympathetic nerve in these cases would appear to be valuable not alone to relieve anginal pains, but also to prevent the occurrence of sudden death.

This therapeutic procedure for angina has never before been carried out.

DAVIS, New York.

TRAITEMENT DU MYXOEDEME (TREATMENT OF MYXEDEMA). SAINTON, Progrès méd. **35**:409 (Sept. 18) 1920.

The only practical mode of administration of thyroid substance today for the amelioration of myxedema is the ingestion of the dried extract. The introduction of glycerin extracts or of thyroid lipoids is dangerous. The administration needs to be discontinuous and varied. During an initial period of adaptation careful recording of the pulse rate, etc., establishes a dosage. During a second period, the patient takes the fixed dose uniformly except for an interruption of a week in each month. During a third period, the dose is diminished and the intervals spaced.

In an adult, when the initial cause of the myxedema is an infection, the dosage of extract will be from 10 to 50 cg. In the myxedema of the infant (3 or 4 years) the dose is 2 to 5 cg.

At the time of puberty, thyroid opotherapy finds aid in the associated use of other endocrinal extracts. Especially in children, calcium, iron, phosphorous or arsenic preparations are useful adjuvants.

Attention is called to the occurrence of a syphilitic myxedema, and in this specific treatment is recommended.

The efficacy of opotherapeutic treatment of myxedema increases in direct ratio with the age at onset.

DAVIS, New York.

ON THE GROWTH OF THE NEURONS COMPOSING THE GASSERIAN GANGLION OF THE ALBINO RAT, BETWEEN BIRTH AND MATURITY. KENJI NITTONO, J. Comp. Neurol. **32**:231 (Oct. 15) 1920.

This paper is a continuation of the studies of the growth of the neurons forming the cranial and spinal ganglions of the rat now in process at the Wistar Institute. The author summarizes his conclusions as follows: The largest cells of the gasserian ganglion show three phases in growth: (1) rapid growth from birth to 20 days, (2) slower growth from 20 to 80 or 100 days, (3) a final phase in which growth is slow, or even a slight atrophy may occur. Growth of the nucleus is slight. Morphologic maturity of the cytoplasm is attained at about 20 days, but the nucelus-plasma ratio, which is high, more than doubles between birth and puberty. Before 80 days the volume of the ganglion cells increases as the area of the head increases. The diameters of the nerve fibers in the root of the fifth nerve are greater than in any of the branches. The fibers grow after the cells have stopped growing. The neurons in the gasserian ganglion differ in various size relations and in the period of growth from those in the ganglion of the seventh cervical nerve. They mature earlier.

HERRICK, Chicago.

PARALYSIS OF SIXTH NERVE WITH OTITIS MEDIA. WILLIAM ZENTMAYER, Am. J. Ophth. **3**:766 (Oct.) 1920

Dr. William Zentmayer presented the following case of Gradenigo's syndrome: C. S., aged 32 years, a milk dealer, had an attack of otitis media beginning Jan. 3, 1920. When seen by George M. Marshall, in consultation with his family physician, Dr. C. B. Schoales, on January 19, there was a copious thick purulent discharge and the upper and posterior wall of the canal was bulging. A simple mastoid operation was done on January 23. The night following the operation the patient had trouble with the left eye and

paralysis of the external rectus was noticed the next morning. On March 8 there was a complete paralysis of the external rectus. The author speaks of the syndrome and points out the situation when it is likely that the nerve is involved. He states that the paralysis may clear up in a few days or at once, following mastoid operation, but it usually persists for weeks or months.

REESE, Philadelphia.

THE INTERMUSCULAR NERVE CELLS OF THE EARTHWORM. A. B. DAWSON, J. Comp. Neurol. **32**:155 (Oct. 15) 1920.

Since the earthworm is so often used as a subject for the study of fundamental physiologic problems and might to advantage more often be so employed, the details of its nervous organization are of general interest. Dawson describes four types of nerve cells of the peripheral nervous plexus in the muscular layers of the body, in addition to the sensory cells of the epidermis. One of these types resembles the latter elements, and these are interpreted as deep-lying sensory cells. The other three types are regarded as motor cells. This arrangement would provide for local peripheral reflexes without participation of the central nervous system, and the author considers the peripheral ganglionic nerve plexus as a survival of an ancestral diffuse nerve net similar to that found in the coelenterates. That is, when the central nervous system was formed by condensation of the primitive diffuse nerve net only a part of the primitive nervous system was so transformed, the remainder persisting as the apparatus here described.

HERRICK, Chicago.

DE LA PHYSIOLOGIE PATHOLOGIQUE ET DU TRAITEMENT DE LA MIGRAINE (PATHOLOGIC PHYSIOLOGY AND TREATMENT OF MIGRAINE). G. DIDSBURY, Progrès méd. **35**:429 (Oct. 2) 1920.

The writer discards toxic, general or even endocrinal theories as an explanation of migraine; he considers that it is caused by a local and superficial condition. In this he follows Nordström. The true cause of migraine must be sought in the presence of localized points of hyperesthesia, and he finds them along the course of the superficial nerves of the neck, cranium or face, and especially at the point of emergence of the nerves. In these locations lesions partaking of the nature of combined subcutaneous cellulitis, a myositis and an interstitial neuritis are present.

The author says that these foci can be cured by massage and believes that when they are disposed of, the migraine does not recur.

DAVIS, New York.

ACCESSORY SINUS DISEASE AND CHOKED DISK. HARVEY CUSHING, J. A. M. A. **75**:237 (July 24) 1920.

In view of the numerous unhappy consequences of indiscriminate and ill-advised operations on the accessory sinuses that have come to the author's attention, he feels obligated to express an opinion on the matter. Infections of the ethmoid cells set up definite types of optic neuritis, but when a choked disk (Stauungs-papilla; papilledema) is present, other factors than a simple inflammation are involved. Evidence of increased intracranial pressure caused either by an inflammatory process within the cranium or by a neoplasm should be considered. The lesion should be treated neurologically instead of by tampering operations on the nose.

ADSON, Rochester, Minn.

Society Transactions

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Meeting, Oct. 21, 1920

EVERETT FLOOD, M.D., *President*

THE INFLUENCE OF THE CEREBRUM ON GROWTH. Presented by DR. A. MYERSON.

This paper dealt with the infantile hemiplegias, the spastic hemiplegias occurring in infancy and in fetal life. It has long been known, though it has not appeared to any great extent in textbooks, that when the cerebrum is injured in fetal life by hemiplegia or inflammation, a limitation of the growth of the side of the body governed by that portion of the cerebrum takes place; the limbs tend to be shortened, the bones to be thinner and the face to be asymmetrical. The ear and the breast are not affected, although in certain cases they are smaller on the involved side. The diameter of the chest is less, and the scapula and clavicle are shorter. Roentgenograms show no particular pathologic phenomena, although they sometimes show that the ossification process seems to lag behind.

Freud's essay on this subject is classic. It is interesting that Freud's first researches should have been in the domain of physical injuries, while his later contributions were on the psychic injuries in fetal life. Perhaps there is a connection beneath the surface.

The phenomena to which this paper calls attention, are first, the contrasting conditions of the hand in adult and infantile hemiplegia. In adult hemiplegia a marked spasticity in the fingers usually develops — the typical hemiplegic hand. In infantile hemiplegia there is hypotonia, which is most marked and constant. This seems worthy of notice first, because the rule in infantile hemiplegias is contrary to the rule in the adult, and second, because it is a constant phenomenon and practically never absent in some degree or other. It is therefore one of the ways of differentiating between infantile and adult hemiplegia.

Another phenomenon concerns the scapula. William Graves of St. Louis has made his life work the study of the condition known as the scaphoid scapula, in which the ordinary convex vertebral border of the scapula is concave in construction, the angle of the spine is changed and the bone itself is smaller and thinner. In his early papers he laid some stress on syphilis as a cause, but now he considers the condition a phenomenon of general pathology. Critics of his work have stated that the shape of the scapula is dependent on use and that the concavity is brought about largely through use. The series of cases which were studied at Dr. Fernald's Institution, Massachusetts School for the Feebleminded, showed that the arm and leg may be completely paralyzed and that there may be no function on that side. The scapulae are also shorter and thinner. They are, however, of the same shape on the affected and the unaffected sides. If the scapula is scaphoid on the sound side, it is the same on the paralyzed side; if the scapula is "normal" on the sound side, it is the same on the affected side. In other words, the shape of the scapula does not depend on use, which is Graves' contention.

OPPORTUNITIES FOR CREATIVE EFFORT BY THE MASSACHUSETTS SOCIETY FOR MENTAL HYGIENE. Presented by Dr. A. W. Stearns.

The Society for Mental Hygiene in this state was one of the first to be organized. It has been an important factor in the development of the National Committee for Mental Hygiene through its membership and its opportunity to help because Massachusetts is so far advanced in these matters. In fact the question has been raised as to whether there is a place in a state so highly organized as Massachusetts for a private society for mental hygiene. This question can be answered in the writer's opinion in the affirmative.

There are many fields of activity which a private society can enter quite apart from those covered by a state organization. However, the excellent development of our state organization makes the function of the Massachusetts Society for Mental Hygiene quite different from that of those in some other states.

The aim of future efforts should be: (1) To create and maintain an enlightened public opinion concerning the relations of mental normality and abnormality to a useful life in the community. Mental disease and psychology are still surrounded with mystery in the minds of the general public. One of the greatest handicaps of mental disease is the attitude which even the most enlightened people take toward it. Efforts at propaganda by state departments often arouse suspicion on the part of the public. There have been occasions in this state during the last few years when resentment has been created by proper attempts of state organizations to stir up public opinion. A private society organized primarily for educational purposes does not meet this opposition. There is little occasion in this state to conduct a campaign concerned primarily with institutional care of the insane, but public opinion is still archaic in its attitude toward the insane or the defective person in the community. It is surprising to find the number of intelligent people who think that many of the insane are kept in institutions by their relatives in order to get their money. The attitude of the public toward an insanity plea in court is hardly intelligent. Again, a failure to recognize the need of adjustment in society for the aged and psychoneurotic leads to broken home ties, divorces and endless litigation. There is evidence to show that the suicide rate can be influenced by propaganda.

(2) To maintain the highest standard in our state institutions. This society should be in an independent position enabling it to commend or criticize when such action is for the best.

(3) To foster research and investigation tending to increase the knowledge of this subject and in that way ultimately to reduce burdens. Every man in this state who develops a new or promising idea, if practical, should feel that this society stands ready to lend its assistance. The routine duties of the state service and the tremendous problem of feeding and housing so many dependents occupy the major part of the time of those officially dealing with the insane. Thus, a society of this sort may well conduct or patronize research.

(4) To extend the investigations now being made in a few centers on the relation between mental disease, personality and crime. There is a tremendous field for mental hygiene in the administration of justice and the practice of law. The good beginnings in this field should be supported and new ones developed.

(5) To take an advanced stand concerning the prevention of feebleminded-ness. The general question as to segregation, sterilization and control of reproduction of the feebleminded can perhaps best be answered and leadership assumed by a private society.

(6) To extend special classes now so successful in a few places. Laws have been passed and state bureaus are now engaged in work with the defective and backward in our schools. The public must be prepared for this innovation by being informed and encouraged to use special classes properly.

(7) To formulate and to carry out organized effort for the care of the handicapped in the community through social service. The last fifty years have been characterized by a tremendous institutional development. The public is now willing to trust the handicapped one to the care of an institution, but further extension is almost prohibited by the question of expense. There are signs everywhere that the contribution for the next generation may be for economical reasons the perfection of community care of the handicapped. Experience has shown that private agencies dealing with a few cases in a personal way can often develop a method more readily and carry on experiments with less risk than can the more cumbersome machinery of a governmental department.

(8) To promote the establishment of courses on mental hygiene in the professional and normal schools. Courses in hygiene are given in many colleges in Massachusetts. These courses have largely to do with the body and should include the mind. Physicians, lawyers and school teachers are continually going forth to their work with too little equipment along these lines. The "urge" to greater effort may well come from our society.

(9) To emphasize the need of mental hygiene in the industries. Individuals succeed or fail according to their mental capacity and ability to adapt themselves to their work. Business houses and industries are employing almost any sort of a person who thinks he can help. This field should be gone over, studied and the fundamentals made matters of routine.

Lastly, there are in this state a great many persons who are professionally or otherwise engaged in work related to mental hygiene. These should be organized, kept informed and asked to advise and so become a greater factor in the progress of our state. Among them are included clergymen, lawyers, doctors, philanthropists, teachers, employment managers, social workers and innumerable others who may be given an opportunity for self-expression through membership in a society for mental hygiene.

IMMIGRATION FROM A MENTAL-HYGIENIC STANDPOINT. Presented by Dr. A. J. Nute.

The earliest migratory movements were due to a class of people known as colonists, as well as immigrants, who sought economic benefit and religious freedom. The character of the migratory movement of a later day has changed so that instead of seeking political rights or religious freedom many are ignorant of the simplest facts regarding the government under which they formerly lived and are apt to confuse the American flag with the dollar bill. The colonist may be classed as an immigrant who was willing to endure the hardships of climate, famine, disease and hostile Indians in order to gain a home and his freedom. The immigrant may do this also, but he is more likely to profit by what the colonist has started. Therefore the present day tendency is to settle in the cities where it is usually possible to live under

conditions more or less satisfactory. In the early settlements selection was the rule; kindred spirits flocked together. Later, as the country grew, the selective element changed and a restrictive policy had to be enforced. The earliest laws passed by the United States, from 1819 to 1882, were largely to protect the immigrant. Since that time laws have been passed largely to protect the United States. In the early days each state had to protect itself as best it could. This was unsatisfactory without national action because only Congress could regulate commerce with foreign countries. New York took the lead and finally Congress was induced to take such steps as would start an organized, uniform immigration policy. At first this was carried out by the states by agreement with the government. In 1890, the federal government assumed control and from that time the laws have gradually tended to exclude the more undesirable types and to protect the desirable.

Traveling in the early days of the sailing ship was a hardship but little appreciated today. The immigrant furnished his own food, and a death rate of 10 per cent. during the voyage was considered normal. From time to time the various countries improved these conditions so that before the days of steamships it was required that immigrants should be provided with certain space, certain food, and some protection in regard to sanitary matters and the competency of ship's officers. Since the invention of steamships the general sanitary and living conditions have improved until at present the so-called steerage passenger travels better than the cabin passenger of one hundred years ago. On arrival at the immigration station means are taken to protect his rights and interests.

In regard to the laws relating to mental inspection, mental defectives have been mandatorily excluded for a comparatively short time. The feebleminded might enter legally up to 1907. Under the laws of 1882 idiots and the insane were the principal mental defectives excluded. In 1893, the law was made more rigid relative to information in regard to arriving aliens. In 1903, epileptics and persons who had been insane within five years and persons who had had two or more attacks of insanity were also barred. In 1907, in addition to the foregoing, a person having a mental defect that might affect his ability to earn a living was excluded, and for the first time a penalty was placed on the steamship company bringing to this country such mental defectives as idiots, imbeciles and epileptics. Provision was also made to deport, at any time within three years after date of entry, any alien that entered the United States in violation of the law or who became a public charge from causes existing prior to landing. The last act of 1917 added constitutional psychopathic invalidity and chronic alcoholism, those having had a previous attack of insanity at any time and persons not coming under the above departments who could be classed as mental defectives. The alien was given the right to appeal when certified to be mentally defective and allowed to present one medical expert in his behalf before a medical board. The penalty or fine for the transportation company was increased, and in addition the deported alien was entitled to a refund from the transportation company of a sum of money equal to that paid by him for transportation from the port of departure to the port of arrival. The provision made to deport any alien was extended from three to five years and the wording changed from "causes existing prior to landing" to "causes existing not affirmatively shown by himself or friends to have arisen subsequent to landing."

The inspection may be likened to a sieve rather than a dam. The immigrant has had at least one and maybe several examinations prior to embarkation. A well-trained examiner can tell at a glance the nationality or race of

the persons who appear before him. This is necessary in order that he may know or recognize the normal from the abnormal. Those detailed are examined in a quiet room after twenty-four hours of rest. Physical examinations are made to make allowance for physical defects. A brief mental examination is given to ascertain the amount of acquired knowledge and to test the mental activities. By this method the normal are released with as little delay as possible. Examinations are then made by at least two doctors on different days, and a certificate is issued when all agree as to the findings. As long as doubt exists reexaminations are made.

DISCUSSION

Dr. Donald Gregg remarked that on shipboard it was extremely interesting to watch immigration inspectors quickly go over two or three hundred people and by simple observation pick out physical and mental defects. He asked Dr. Nute to mention more specifically the general signs that an inspector would look for in examining several hundred men.

Dr. A. W. Stearns objected to the inference that we were all immigrants. The publicity that has been given during the last year to the early history of this country would tend to show that this is not so. Several persons have attempted (Dr. James J. Putnam in particular) to define or describe the New England character. The population of New England was not an immigrant population but grew through reproduction. For a period of twenty years about 20,000 people came to this country and then, with the exception of the Scotch Irish, immigration stopped. As many returned to Europe as came so that for nearly 200 years the increase of population was by reproduction. This was also accompanied by extreme isolation. There were no large cities, and the settlements of colonists were widely separated so that a type grew up which has become quite distinct from the immigrant type.

Dr. Stearns also suggested that great differences of mentality in children and illiterates could be detected by skilled observation of the facial expression and manner. He has found this to be true in his experience with the lowest type of Southern negro.

Dr. A. J. Nute, in answer to Dr. Stearns, said that it was true that facial expression was a guide to intelligence. For instance, if an intelligent immigrant child of about 2 years is brought into a room hung with bright flags his passive expression at once will change to an animated one. A simple toy will give the same result.

In reply to Dr. Gregg's question, Dr. Nute said that an experienced inspector must be familiar with the types of people he examines. He must be able to tell at a glance the nationality of the man passing before him; if he cannot do this, he is not a competent examiner. The stolidity which is to be expected in a Pole, for example, might be indicative of a dementia in an Italian. If an Italian woman had the flush of a Scandinavian, she would be taken aside in order to discover whether she had a temperature. Again, the immigrant dresses in his best when he comes into port. He wants to make the best showing he can. If one comes along carelessly dressed and appearing indifferent, it immediately attracts attention and it is well to investigate the mental attitude of that person. Now the gross defects are fairly well eliminated on the ship, and there is time to devote to the finer defects. This fact is responsible for the large increase in the detection of the mentally defective.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Oct. 22, 1920

SAMUEL D. INGHAM, M.D., *President*

A CASE OF EPIDEMIC ENCEPHALITIS OF THE PARALYSIS AGITANS TYPE. Presented by DR. F. H. LEAVITT.

Dr. Leavitt said that the case was of interest because the spinal fluid examination showed a positive Wassermann reaction, a cell count of 22, a positive globulin reaction, and a colloidal gold curve of 3455432110, while the blood Wassermann reaction was negative.

The previous history was negative. The patient was 31 years old and married. Feb. 21, 1920, his temperature was 103 F., which was followed by a general "neuritis," with severe pains in all parts of the body and some delirium for two weeks. He had diplopia for weeks. He had incontinence of urine and feces for seven weeks. A trophic bed sore developed after five weeks, which did not heal. Gradually a deformity of the feet developed, which prevented the patient from walking. The pain disappeared entirely in three weeks and was followed by tremor of the hands and monotonous speech. A little later tremor was noticeable in the legs, and a paralysis agitans picture developed and has persisted ever since.

There was constant tremor of the toes of the left foot and of both arms, more marked on the left and accentuated by emotion and voluntary movements. The facies were mask-like and expressionless, and the speech was monotonous and tremulous. He could move all muscles of the face voluntarily but to a limited degree. There was no difficulty in deglutition or phonation. All movements of the extremities were weak and slowly and awkwardly performed. Both feet were held in the position of talipes equino-varus. Stroking the sole of the foot caused withdrawal of the entire leg but no movement of the toes. There was no ankle clonus. The knee jerks were equal but weak. Cremasteric and abdominal reflexes were normal. Sensibility to touch, heat, cold and pain was normal. There was a large sloughing bed sore over the sacrum. The sphincters were controlled. The results of ocular examination were negative. Hearing, vision and taste were normal.

During his stay in the hospital, otitis media with peripheral cellulitis, caused by a streptococcus pyogenes infection, developed, which subsided two days after the drum was incised.

After treatment by mercurial inunctions and potassium iodid in small doses, the blood and spinal fluid Wassermann reactions were negative; cell count, 2; globulin +; colloidal gold 1111112100.

In the majority of cases of epidemic encephalitis the spinal fluid reaction has been negative, but in this patient it was positive, and it has not been proved that the encephalitis could not cause the reaction. Furthermore, the patient did not improve under antisyphilitic treatment, but rapidly and progressively became worse. Neurosyphilis does not produce a clinical picture simulating paralysis agitans as did the picture in this case.

DISCUSSION

DR. CHARLES W. BURR said that the name lethargic encephalitis was not used because he believed that it was misleading since many of the patients never had lethargy. Epidemic encephalitis is a better designation. Instead

of lethargy, the patient may be delirious or somnolent during the day and delirious at night. When Dr. Burr first saw this man in bed and saw only his face, he thought it was a case of paralysis agitans. At that time Dr. Burr knew nothing about his history. Then the patient had the same tremor in his hand that he now has in his foot. It was not surprising that the spinal fluid Wassermann reaction was negative under treatment. Dr. Burr wondered whether encephalitis could make a latent syphilis active, just as in childbirth a latent malaria in the mother may be made active, or whether in encephalitis apart from syphilis there may be a positive Wassermann reaction due to the encephalitic infection itself.

DR. JOHN H. W. RHEIN said that the spinal fluid examination had been made in a large number of cases reported in the literature, and a negative Wassermann reaction had been found in all instances. In one case which he reported, there was a positive spinal fluid Wassermann reaction but the patient died and no necropsy examination was made. He regarded the rare occurrence of a positive Wassermann reaction in epidemic encephalitis in the spinal fluid as a coincidence.

TWO BOYS, BROTHERS, WITH CHOREIC ATAXIC MOVEMENTS.

Presented by DR. J. HENDRIE LLOYD.

Dr. Lloyd said these cases might perhaps be instances of atypical Friedreich's ataxia, although he was not sure that this term was the proper designation for them. The father and mother were well and had had eight children, of whom the other six were well. The patients did not come next to each other in order of birth, there having been two healthy children between them. There was no family history of any such disease on either side.

The condition of the elder brother, 20 years old, began with irregular movements when he was 4 years of age. These had greatly increased during the last three years, so that he was almost unable to walk. His gait was rather reeling or staggering, not unlike a cerebellar gait. He had free, even increased, knee jerks, but the ankle reflexes were diminished and there was no Babinski reflex. There was vertical nystagmus; the eyegrounds were normal. The pupils reacted to light and on accommodation. There was a speech defect, not typical of Friedreich's ataxia, and the boy was rather dull in mind. There was no pain or sensory disturbance. As he sat there was a slight scoliosis. The Bárány test (made by Dr. Hunter) showed that the impulses went through all pathways, but after rotation the nystagmus was of unusual duration (48 to 57 seconds). Past pointing, falling and hearing were normal. The Wassermann tests of the blood and spinal fluid were negative.

The younger brother, 14 years old, had movements that were rather more choreiform than those of his brother. They involved the face as well as the limbs, and dated from an early age, not accurately stated. He was able to walk without difficulty. He also had a slight speech defect, little, if any, nystagmus, and the pupils reacted normally. The Bárány tests showed rather prolonged nystagmus after rotation. There was some past pointing. The knee reflexes were present; the right was decreased.

The question is whether there is a cerebellar lesion in these cases, or whether they are of a purely spinal type. If the former, they would probably approach Marie's cerebellar ataxia. The preserved, even increased, knee jerks rather point against Friedreich's ataxia, although some such cases have been reported.

DISCUSSION

DR. F. X. DERCUM said he had shown a patient before the Neurological Society about three years ago, a man of 28, whose symptoms, beginning at 6 years of age, were very much like those of the elder of the two boys shown by Dr. Lloyd. Dr. Dercum had regarded the case as in all probability one of hereditary cerebellar ataxia. There was a marked family history. Dr. Dercum thought it quite likely that heredocerebellar ataxia and Friedreich's ataxia were closely related conditions, as in both it would seem that there are feebleness of development and consequent degenerations, essentially biologic in nature. (Dr. Dercum's case was published in the *New York Medical Journal*, Aug. 25, 1917.)

DR. JOHN H. W. RHEIN said that some years ago he had had an opportunity to study a family group with spastic paralysis. At that time he went over the entire literature of family groups with conditions of the spinal type. It seemed to him at that time that there was great confusion in the classification of these diseases and it appeared possible to group these cases so that at one end could be placed typical Friedreich's ataxia and at the other, typical spastic paraplegias with several different types, more or less sharply defined, in between. Between these two typical groups, there were cases in which the arms were involved; others in which the cerebellar symptoms were added; still others exhibiting bulbar symptoms; another group showed muscular atrophy; another group had symptoms that resembled those of disseminated sclerosis. It seemed to him that these groups were mere subdivisions of the same disease, and all represented probably the same pathologic process.

DR. WILLIAM G. SPILLER said that while these cases presented some features atypical of Friedreich's ataxia, he thought it a mistake to discard, or to put in a class apart, certain cases of this disease in which a few uncommon symptoms occurred. Friedreich's ataxia is capable of many variations; it is a posterolateral sclerosis, possibly the best example of a true systemic combined sclerosis, and the symptoms vary as the lesions predominate in one system of fibers or another. The degree of implication of the cerebellum in this disease has been a matter of dispute during many years. While it is desirable to recognize the distinctions between similar diseases of the nervous system, it is equally important to recognize the connecting links.

DR. T. H. WEISENBURG said that Dr. Lloyd had given him the opportunity to study his cases before their presentation, and he believed that they were distinctly cerebellar and that the pathology consisted either in a congenital lack of development or atrophy in a definite part of the cerebellar system. The question had come up as to whether these cases could not be diagnosed either as Friedreich's ataxia or whether they belonged to Marie's hereditary cerebellar ataxia. Regarding Marie's type, he called attention to the fact that when Marie first described this disease in 1893, he based it entirely on the observations of others.

Analysis of the clinical records of these cases, as pointed out by Gordon Holmes, shows that they were by no means identical or even similar, and pathologic studies showed that in only one case, that of Fraser, was there evidence of acquired cerebellar disease. Dr. Weisenburg agreed with Holmes that no form of disease existed to which the term hereditary cerebellar ataxia could be aptly applied, and said it was apparent that this title has been a convenient pigeon-hole in which every obscure or even plain type of cerebellar disease has been placed.

There will never be progress in cerebellar diagnosis so long as neurologists persist in using the old time classification and symptomatology. Most modern reports of cerebellar cases are incomplete, and no attempt has been made to delimit cerebellar asynergy to parts of the body. The description of most cerebellar cases gives one the idea that asynergy and adiadokocinesis are merely symptoms, whereas every one should know by this time that the primary attribute of the cerebellum is to synergize all motor functions and whatever symptoms occur are the result of loss of synergic action of certain muscles concerned in a definite movement.

Dr. J. Hendrie Lloyd said that the discussion had brought out just what he wanted. He agreed that the term Friedreich's ataxia admits of some variety. In Griffith's paper, written about thirty years ago, including most of the cases reported up to that time, a few were reported with exaggerated knee jerks; but not all the cases in that paper had been reported by competent observers. In Friedreich's ataxia a Babinski reflex had sometimes been observed; he had seen one such case. Marie's cerebellar ataxia had always seemed to him to be a rather different disease, nevertheless it could not be ignored in considering the present cases. The elder boy especially suggested the possibility of a cerebellar lesion. He agreed with Dr. Weisenburg that this whole subject of the familial ataxias needs careful revision. The members of the society would probably recall that less than a year ago Dr. Lloyd had shown two colored boys, brothers, with Friedreich's ataxia. One of these boys had since died of pneumonia, and sections from the cerebellum and cord will soon be ready for examination.

PROVOCATIVE SPINAL FLUID CHANGES IN NEUROSYPHILIS.

Presented by Drs. H. C. Solomon, Boston, and J. V. Klauder.

The provocative arsphenamin reaction in the blood serum is a familiar phenomenon. In many clinics when a patient suspected of having syphilis gives a negative Wassermann reaction on the blood serum, an injection of arsphenamin is given, and then the blood is tested at daily intervals thereafter, because it apparently has been shown in many cases of this sort that after the injection of arsphenamin the Wassermann reaction will be positive.

The purpose of this paper was to call attention to a similar reaction taking place in the spinal fluid. Case histories were presented in which after either intravenous or intraspinal injections, a negative spinal fluid became positive, or the positive pathologic findings were intensified by such treatment. From the clinical standpoint, intensification of symptoms relating to the central nervous system has been frequently described as occurring after treatment. To this phenomenon the term neurorecidive or neurorecurrence has been applied. One is not sure in these cases that the resulting intensification of symptoms is due to the introduction of the drug, or that it is a mere coincidence that an increase of symptoms appears following the injection. However, in secondary syphilis the intensification of symptoms following arsphenamin injection has occurred so frequently that it has been considered as due to liberation of toxins resulting from the lytic action of arsphenamin on spirochetes. This is the so-called Herxheimer reaction. If this can occur with skin lesions, there is every reason to suppose that the same might occur in neurosyphilis. However this may be, the following cases would seem to show that following intravenous and intraspinal injections of arsphenamin or arsphenamized serum, the spinal fluid findings may become more strongly positive.

These cases illustrate the fact that after antisyphilitic treatment a negative spinal fluid may become positive, or one that is slightly positive may give more strongly positive reactions.

In Case 1 the diagnosis was cleared by the effect of a provocative injection of arsphenamin. In this case the spinal fluid, which was negative prior to antisyphilitic treatment, became strongly positive in all tests after one injection of arsphenamin. It is worth while to note that the blood did not become positive. This result is also found in the other cases of this series.

Case 2 of the series shows the intensification of the spinal fluid Wassermann reaction after intravenous injection and also the provocation of a positive colloidal gold reaction, globulin and pleocytosis. It is perhaps of interest to point out in this discussion the curious fact that no provocative change was noted in the serum Wassermann reaction. In the light of present knowledge, perhaps, one can conclude that the patient is cured so far as visceral involvement is concerned. Latent foci of spirochetes in the nervous system apparently were activated by treatment, as was manifested by the provocative spinal fluid changes.

The patient in Case 3 presented so definite a picture of tabes that no doubt of the diagnosis could exist, despite a spinal fluid that was negative in all phases, with the exception of a small amount of globulin and albumin increase. No provocative result occurred either on the blood or spinal fluid as the result of intravenous injection, but intraspinal subdural injections of arsphenamized serum provoked positive Wassermann and gold reactions. It is worth while to note that under treatment these reactions were reduced to almost normal. From the clinical standpoint, improvement took place even during the period in which the tests became more strongly positive. The patient, who was in the paralytic state of tabes, being confined to bed, became able to walk without a cane, although quite ataxic.

Case 4 was that of a patient with undoubted tabetic disease, showing negative blood and spinal fluid. Under intraspinal injections the spinal fluid showed positive globulin, albumin, pleocytosis and gold reactions, but the blood and spinal fluid Wassermann reactions remained negative. Contrary to the experience in Case 3, no improvement in the patient resulted from this treatment.

Case 5 was that of another patient in whom the clinical evidence made a diagnosis of tabes certain, but whose spinal fluid Wassermann reaction was negative. The blood, however, was positive, and there were 139 cells per c.mm. in the fluid. After intravenous injections of arsphenamin, the Wassermann reaction in the blood serum became negative while the spinal fluid Wassermann reaction became positive. The cell count decreased, but the colloidal gold reaction became more strongly positive. From the clinical standpoint, the result of treatment was eminently satisfactory, as all the pain disappeared, the ataxia became markedly improved and the patient was able to return to work after having been incapacitated for some time.

Case 6 showed the provocative results in the ventricular fluid after intraventricular injections, similar to that observed in the spinal fluid of the preceding cases.

Altman and Dreyfus have shown that arsphenamin may have a provocative influence on the spinal fluid in cases of primary and secondary syphilis. These writers speak of this phenomenon as a provocative neurorecidive. A criticism of this conclusion might be offered, as it is quite possible that the condition had nothing to do with the introduction of arsphenamin, but might have

occurred had this not been done. As this occurred some time after the injection, and as there was only one injection, this case cannot be considered as strong evidence, but as illustrative of this type of reaction.

A study similar to that of Altman and Dreyfus was made by Dr. Klauder. In a series of twenty-five cases of secondary syphilis, the spinal fluid was examined before and after a course of four intravenous injections of arsphenamin, administered at intervals of from seven to ten days. Provocative spinal fluid changes were observed in some of the cases on the second examination of the spinal fluid. This observation and those of Altman and Dreyfus are in accord with the hypothesis of Gennerich that arsphenamin is capable of producing provocative spinal fluid changes in the early period of syphilis.

The change of a spinal fluid by arsphenamin therapy from negative to positive is, in all probability, the expression of a Herxheimer reaction. A neurorecidive or neurorecurrence following arsphenamin injection is probably a clinical expression of the same reaction. The apparent provocative reactions in the spinal fluid of neurosyphilitic patients, as herein reported, would seem to be of a similar nature and in line with the above. It should be emphasized that this provocative reaction is not a frequent occurrence, even in cases in which there is evidence of central nervous system involvement with negative findings in the spinal fluid.

Case histories of neurosyphilis were presented in which no provocative spinal change was noted following treatment with arsphenamin.

A note was made of cases of neurosyphilis which were made worse by arsphenamin therapy. These reactions doubtless manifest the powerful spirocheticidal action of the drug. The arsphenamin either activates foci of spirochetes or, as a result of the lysis of great masses of organisms, the toxins are liberated in large amounts, which produces pathologic results. We may, then, assume that this result is expressed in a laboratory way by the appearance of positive reactions in the blood and spinal fluid, and in a clinical way by the appearance of neurorecidives and the accentuation of symptoms.

DISCUSSION

Dr. H. C. Solomon, Boston, said that he would like to say a word in relation to the effect of arsenic in the production of neurorecidives. For a time there was in Boston general acceptance of the idea that arsphenamin produced deafness and blindness. Dr. Crockett of the Massachusetts Charity Eye and Ear Infirmary made a study of syphilitic nerve deafness before the arsphenamin era and after. He found the incidence was essentially the same after the introduction of arsphenamin, if anything a little less, and Dr. Solomon agreed with this conclusion. For the past year and a half he had in a sense dropped the rôle of neurologist and had become a syphilologist in order to learn what he could about these problems.

In the Massachusetts General Hospital it was the rule to put every syphilitic patient on arsphenamin at once. This treatment was followed up by mercury. They did not find that many of the patients developed nervous system lesions after treatment was begun. Dr. Solomon said he had seen many patients with secondary syphilis, untreated, who had thrombosis of various cerebral vessels occurring within three to six months. He saw an occasional patient who had received treatment and developed cerebrospinal involvement during the secondary period.

It was obvious that half a dozen injections of arsphenamin would not help a paretic. Six years ago Dr. Solomon began to give from 0.6 to 0.9 gm. of

arsphenamin twice a week over a period of from six months to a year. He had patients now who began to receive treatment six years ago and, contrary to the usual run of paretic patients, they are now happily at work. It surprised him that they had even not had kidney involvement. In many ways arsphenamin is a dangerous drug and should be used with care by those who are inexperienced in its use. Medical examiners in Boston gave a warning, "You would better not treat patients at your office unless you know what you are doing." Sudden deaths have occurred from the use of arsphenamin. Of course the same thing is true in surgery.

Dr. J. Hendrie Lloyd said that this was a most important subject. In the last few years he had seen in his service in the Philadelphia General Hospital four cases of deafness in early syphilis following the use of this arsenical drug. In two of them there was associated paralysis of the seventh nerve. In all cases deafness had occurred within a year of the primary lesion. He had been on the Philadelphia General Hospital staff for a great many years, and he had never seen such conditions until the introduction of this arsenical preparation. He only stated this for what it was worth. He showed two of these cases before this Society a year or two ago. Dr. Lloyd said the question arises "Are we doing this with our drug, or are we not?" It is one of the worst complications that can come in syphilis. Involvement of the eighth nerves causes total bilateral deafness and is incurable.

Dr. Weisenburg said that there was being developed in Philadelphia, and perhaps all over the country, a group of chronic arsphenamin takers who went from one clinic to another for injections. For example, in the Polyclinic Hospital, which has one of the largest syphilitic clinics in the country, there were quite a number of tabetic patients who came weekly for the injections of arsphenamin. A number had had over a hundred injections. These patients have the idea that the only method of cure is to receive weekly arsphenamin injections, and if one clinic refuses to give it to them, they go to another, frequently not telling the truth about how many injections they have received. Dr. Weisenburg was impressed with the ease with which these patients took the injections. They came practically without any preparation; after receiving the injection they left promptly, and often went to work directly after, without difficulty.

A CASE OF BRAIN ABSCESS CAUSED BY A POCKET KNIFE INJURY OF THE SKULL. Presented by Dr. H. F. Dunlap.

History.—A colored man, aged 19, admitted to the Philadelphia General Hospital, Sept. 11, 1920, complained of severe headache, pain through his eyes and stiffness of the neck. His past history was negative except for an injury to the left side of the head from an alleged razor cut received in a fight two years before. There were no sequelae. He denied venereal disease but admitted excessive alcoholic indulgence. The present illness began three weeks ago with severe and continuous generalized headache, dull aching pain in the eyes and stiffness in the neck and shoulders beginning one week later. Since that time he had had from twelve to twenty-four hour intervals of inability to talk or remember things but he had never become unconscious. There had been no vomiting or convulsions. This condition obtained at the time of his admission.

Physical Examination.—The pupils were unequal, the right being larger than the left, but they reacted well. The left palpebral fissure was narrower. There

was paralysis of both external recti, more marked on the left. Convergence was normal. There was bilateral choked disk of 4 diopters, with intense engorgement of the veins, a few white spots on the retina and a few scattered hemorrhages. The left brow showed slight paresis. Speech was slow, stammering and indecisive. The neck was markedly rigid. The tendon reflexes were all present and active; superficial reflexes were normal. No Kernig's sign and no ataxia were present. Sensation could not be tested on account of the patient's mental condition. He was lethargic and somewhat stuporous, although he could be aroused. He showed restlessness and irritability but slept a great deal. The blood pressure was systolic, 120; diastolic, 70. Leukocytosis ranged from 9,800 to 10,800 with 78 per cent. of polymorphonuclears. The blood Wassermann test and blood culture were negative. The urine examination was negative. A daily spinal fluid examination was made. The spinal fluid was always clear until the last specimen. The cell count ranged from 730 to 840, with the exception of the last specimen, which was 2,000 plus. The cells were mostly lymphocytes—from 84 to 93 per cent., with the exception of the last specimen which showed a strongly polymorphonuclear leukocytosis. The spinal fluid was bacteriologically negative, with the exception of the last specimen which showed *Micrococcus tetragenus* and later a staphylococcus. The globulin tests were strongly positive, though the Wassermann and Fehling tests were negative. The temperature ranged from normal to 101, and the pulse from 50 to 60 until the last three days when it mounted as high as 100. The patient was unable to cooperate and too sick to permit a roentgen-ray examination of his head.

Progress: The headaches persisted, although they were relieved by lumbar puncture to a certain extent. At times he showed a rather profound stupor, but on the third and fourth days he was more alert and even said his head was better, although his neck and shoulders were more rigid. On the fifth day he became disoriented, and the following day he was restless and delirious. He died on the sixth day after admission.

Postmortem Findings: The point of a pen knife, 2 cm. long by 5 mm. wide, projected from the inner surface of the calvarium at the left parieto-occipital fissure, extending down through the dura and into the brain substance. Thick creamy pus exuded from this point. The dura was markedly thickened and adherent here, both to the brain and the calvarium. In the left parietal lobe, just above the sylvian fissure and bounded anteriorly by the postcentral gyrus, above by the great longitudinal fissure and posteriorly by the anterior portion of the occipital lobe, was an abscess 4 cm. in diameter, whose external wall was the thickened dura, which formed a pyogenic membrane. The surrounding cerebral tissue was soft, and there was no sharp demarcation between it and the healthy tissue. The lateral ventricles contained a small amount of pus which on culture proved sterile. There was no exudate over the general cortical surface. Cultures from the abscess revealed *Staphylococcus aureus* and *Micrococcus tetragenus*.

DISCUSSION

Dr. Cadwalader said this case showed that an abscess may develop long after the injury to the brain has been received. In such cases lumbar puncture may be useful to determine whether or not a diffuse meningitis is developing. A leukocytosis of the spinal fluid would indicate such a complication; and if it is present, the case is not a favorable one for operation. Meningitis often develops in such cases by the extension of the infection from the site of injury to the lateral ventricle of the brain.

Dr. T. H. Weisenburg said that when the patient was admitted, he presented many of the symptoms of lethargic encephalitis, and this diagnosis was tentatively made because of the stupor, sixth nerve palsy and the limb symptoms.

The presence of the piece of pocket-knife, which was found on stripping the dura from the calvarium, was not suspected. It is extraordinary that this should have been within his skull for two years without causing any symptoms previous to two weeks of his entrance to the hospital.

EXTRADURAL ABSCESS OF THE MIDTHORACIC REGION OF THE SPINAL CANAL SECONDARY TO A BOIL IN THE NECK. Presented by Dr. William G. Spiller and Dr. V. W. M. Wright.

J. H., a white man, 36 years of age, was admitted to the Philadelphia General Hospital, Sept. 29, 1920. He began to have pains in his back August 28, over what he termed the kidney region. Up to this time he had enjoyed good health. He had chills and fever, which did not last long. He denied having had any injury to the spine or hip except a blow on his hip during August. About July 15, 1920, he had had a large carbuncle on the left side of his neck in its upper part, also two over the sacrum, one on the right upper limb and two on the left upper limb. He had slight stiffness in his back, following the blow to the hip, and severe pain over the lumbar vertebrae. This pain, which developed with chills, was severe; his back was so stiff that he could bend forward only with difficulty. The pain radiated up and down the lumbar region and anteriorly through the abdomen, and as it subsided it left the affected parts "dead." The pains then began to spread down the lower limbs and through the chest and by September 4 they were very severe. He described the pains as "thunder and lightning"; they came like a flash and felt like "fire and needles." They ceased on the twenty-fifth day (September 22). Paralysis then developed in the lower limbs. During the day of September 25, he walked normally, but during the night he arose from his bed at 10 p. m. and found he had difficulty in walking. Shortly after midnight he was unable to move either limb, and sensation in the lower limbs was lost. At the same time he lost control of his rectal and vesical sphincters and became incontinent. The next day he had to be catheterized. Since that time he has remained in the same condition.

September 30, 3 p. m.: A spinal puncture was made in the lumbar region and 10 c.c. of a greenish-yellow fluid were removed. It ran slowly, and before it could be carried to the laboratory it was two-thirds coagulated; when it reached the laboratory it was entirely coagulated. Examination showed a heavy trace of globulin, 30 cells to the cm., equal parts of lymphocytes and leukocytes and no bacilli or other organism. The spinal Wassermann reaction was $++++$ in cholesterin. The blood Wassermann reaction was negative. Hemoglobin was 90 per cent.; red blood cells, 4,040,000; white blood cells, 31,200. Roentgen-ray examination revealed nothing of importance.

The face and upper limbs were not affected. Complete anesthesia for all forms of sensation was present anteriorly and posteriorly from about the seventh intercostal space down, including the lower limbs. A line drawn around the trunk at the lower part of the ensiform process gave a zone of hyperalgesia to pin prick. The lower limbs were flaccid and the tendon reflexes of these limbs were lost. Control of bladder and rectum was lost, and a bed sore developed over the sacrum.

On account of the history of a severe boil on the neck in July and xanthochromia of the spinal fluid with signs of a transverse lesion of the spinal cord at about the seventh thoracic root, it seemed evident that some lesion had occluded the dural canal at this level causing the xanthochromia and pressure on the cord, and producing the signs of complete transverse lesion. The severe boil of the neck made the diagnosis of extradural abscess from this boil possible.

A lumbar puncture was made at the tip of the spinous process of the sixth thoracic vertebra, in order to enter the intercostal foramen between the seventh and eighth thoracic vertebrae, and about 6 to 8 c.c. of pus were aspirated at this level. This pus was extradural. Stained preparations showed a staphylococcus. Culture showed pure culture of *Staphylococcus aureus*. The patient was transferred immediately to the surgical wards, and Dr. T. T. Thomas performed a laminectomy at midnight and removed about 2⅔ drams of pus.

Under autogenous vaccines the leukocyte count of the blood was greatly reduced.

Dr. Spiller, in closing, said that this was at least the third case of this character he had seen, and the subject seemed to be important. The man had the symptoms of a transverse lesion. The lumbar puncture which had been performed showed the Nonne-Froin syndrome. There was clearly obstruction to the circulation of the cerebrospinal fluid. Dr. Spiller believed that pus had entered the vertebral column and gone downward until it met some resistance, and had caused an abscess at a lower level on the outside of the dura but within the vertebral column. There was evidently pressure on the cord, but the lumbar puncture had shown that there was no pus within the spinal dura at the time of the puncture.

NOTE.—Death occurred Oct. 28, 1920. The necropsy performed after the meeting of the society showed that a severe purulent meningitis of the pia of the brain had developed; this probably was of late date. There was also a localized empyema of the right side.

DISCUSSION

DR. T. H. WEISENBURG said that six years ago he had had under his observation a man 67 years of age who had all the symptoms of a spinal cord tumor at the level of the left second, third, fourth and fifth lumbar segments. In addition to pain and diminution of power in both limbs, the patient had also increase of knee jerk on the right, with absence of it on the left. The Achilles' reflex was increased on both sides, and a bilateral Babinski reflex was present. The cremasteric reflex was lost on the left side. Lumbar puncture was made in the second lumbar interspace. It was very painful, and the needle encountered considerable resistance. The amount of fluid removed was considerable, but there were too many blood cells to allow conclusions. Within a week the pain and weakness in the limbs became less, and when he left for home, about a month later, the left knee jerk and cremasteric reflex had returned somewhat and the Babinski reflexes were not so marked. This patient had been kept under constant observation for a period of four years. In a personal communication from his son within a few months, the patient was reported as apparently in good health with no organic symptoms. Since that experience, Dr. Weisenburg had advised and performed punctures at the site of all lesions at which tumors were suspected.

BRACHIAL PARALYSIS FROM THROMBOSIS OF THE SUBCLAVIAN VEIN WITH THE REPORT OF TWO CASES. Presented by Dr. George Wilson.

Dr. Wilson reported the cases for two reasons: first, because of the great rarity of thrombosis of the large veins draining the upper extremities, and second, because one of the patients was thought for a time to have a cerebral monoplegia.

The first case was that of a negro, 25 years of age, admitted to the dispensary for nervous diseases of the Hospital of the University of Pennsylvania, April 12, 1920, and referred to the house the next day. His chief complaint was swelling and paralysis of the right upper extremity. The man stated that he was well until April 12, 1920. On that day he took a nap on a pile of coal ashes still a little warm. He awoke three hours later with numbness in the right arm. He noticed that the entire arm was swollen and that on the extensor surface of the forearm there were several large blisters. He denied syphilis.

At the first examination, because of a suspected weakness of the lower half of the right side of the face, a cerebral thrombosis was considered. The eyes, cranial nerves, heart and abdomen were normal. There was slight enlargement of the posterior cervical glands. The lungs presented evidence of acute bronchitis with infiltration of the right upper lobe. The right upper extremity was greatly swollen from the hand to the shoulder. The swelling pitted on pressure. Radial and brachial pulses could not be felt. On the extensor surface of the forearm and arm were several large blebs. The tendon reflexes were absent and sensation for touch, pain and temperature were distinctly impaired. The entire extremity was completely paralyzed. The other extremities were normal. The sputum, repeatedly examined, was negative for tubercle bacilli. The blood showed a mild secondary anemia with the leukocytes continually below 10,000. A blood culture was sterile. The Wassermann reaction was delayed negative. The urine was negative except toward the end when evidence of nephritis was present. A guinea-pig was injected with a centrifuged sediment of sputum and died from tuberculosis.

The patient recovered some power in the hand and forearm, and the edema disappeared to a large degree. The radial pulse returned with the receding edema. The patient was transferred to the medical service, May 13, 1920.

The diagnosis made was brachial palsy due to a thrombophlebitis of the subclavian vein. The lung condition was considered tuberculous. Death occurred, June 6, 1920. Necropsy examination showed miliary tuberculosis. The mediastinal, cervical and retroperitoneal lymph nodes were caseous. It was unfortunate that the subclavian vein was not dissected out at necropsy.

The second patient shown to the society was a negro, 22 years of age, single, a lead worker. He was admitted to the Philadelphia General Hospital, Sept. 16, 1920, in Dr. Spiller's service. The patient had been admitted to the same hospital three times before; the first time with secondary syphilis, the second time with a bubo and the third time with tertiary syphilis. He worked with lead for some time but quit because of symptoms suggesting lead poisoning.

On July 31, 1920, he worked and went to bed feeling well. He awoke the following morning and found that he was lying on the left arm, that the entire extremity was intensely swollen and that it was paralyzed. He had no pain for the first twenty-four hours, but had much pain after that. He went to a hospital where about thirty incisions were made with the discharge of considerable serum. The swelling subsided to some extent.

The patient was of a low mental type. The eyes, ears, cranial nerves, lungs, heart and abdomen were normal. The left upper extremity was completely paralyzed with lost reflexes and great impairment of sensation from the elbow down. Many scars were present on the arm. The swelling had entirely subsided. There was considerable atrophy of the hand and forearm. Examination of the urine showed lead. Blood examination revealed a secondary anemia with granular degeneration of the red cells. The Wassermann reaction was negative. The diagnosis in this case was the same as in Case 1. In this case the etiologic factor was syphilis, whereas in the first case the cause was tuberculosis.

A considerable degree of recovery has taken place, probably influenced greatly by the early incisions. The patient is on antisyphilitic treatment and is receiving massage and galvanism.

The paralysis in the cases reported was considered due to the pressure of an enlarged and thrombosed subclavian vein on the brachial plexus.

DISCUSSION

DR. CADWALADER said Dr. Wilson's case showed that the brachial plexus could be paralyzed by the pressure exerted on it by a thrombus in the subclavian vein. It was not uncommon to find cases of gunshot wound, in France, in which a hematoma had formed, or a thrombus that compressed the plexus and caused paralysis. When removed or absorbed, the paralysis disappeared.

A CASE OF TABES WITH UNUSUAL SYMPTOMS. Presented by DR. ALFRED GORDON.

A man, 54 years of age, was a typical tabetic. His disease was of many years' standing. About three months before, he developed sharp shooting pains in the midcervical region, radiating laterally. At the same time it was observed that the inner border and lower angle of the left scapula were receding from the thorax when the scapula was at rest. Since the function of the rhomboid muscle is to hold the scapula against the thorax in opposition to the teres major and serratus muscles, it is evident that the left rhomboid was paralyzed. The nerve supply of the latter comes from the fourth and fifth cervical segments. The muscular paralysis and the pain in the midcervical region are indications of a probable extension of the tabetic process to the cervical cord and its meninges.

During the last two years the patient has been suffering also from marked ptyalism. The latter was so pronounced that it interfered with sleep. The mouth was always filled with mucus, and the dribbling of saliva was constant. At night the pillow was saturated with it. The involvement of the parotid, submaxillary and sublingual glands was manifest. Their nerve supply comes principally from the sympathetic system. There is, therefore, a sympathetic complication of tabes. Since the amount of mercury used by the patient was extremely small—for the last two years not any—mercury cannot be incriminated in the excessive salivation. The history shows that no other internal factor was the cause of the distressing condition. The condition was probably a tabetic complication.

Book Reviews

DAS WESEN DER PSYCHIATRISCHEN ERKENNTNIS. By Arthur Kronfeld, M.D. Pp. 482. Berlin: Springer, 1920.

Not long before the war began, a group of men arose in Germany who were interested in correlating the material of psychopathology with philosophical conceptions. The journal of this group was the *Zeitschrift für Pathopsychologie*. Of this group, Kronfeld has been a conspicuous representative, and this volume initiates a comprehensive treatment of problems presented in this field.

A facile but none the less objective touchstone of a book's content is the index of names. Clearer evidence of the volume's tendency could hardly be offered, within the space limits, than the comparative numbers of times the following writers are indexed: Abderhalden 2, Bergson 12, Bleuler 27, Brentano 42, Brodmann 1, Flechsig 1, Freud 21, Fries 50, Gaupp 1, Griesinger 16, Gudden 1, Hegel 10, Heinroth 10, Husserl 45 (Janet 0), Jung 2, Kant 86, Kraepelin 19, Lipps 47, Monakow 1, Nelson 35, Nissl 2, Pinel 1, Rickert 47, Sommer 3, Wernicke 7, and Wundt 24.

Let this testify to the essentially philosophical orientation of the work, which is so pronounced as to suggest that the psychopathologist should leave any but the most general criticism to someone more practiced in dealing with philosophical concepts. It is hard to see that the practicing psychiatrist is more concerned with it than the geologist with the headstones of a necropolis. Dr. Kronfeld has reared an imposing mausoleum, but most readers of this journal will find the interior tenebrous.

The author gives earnest of more concrete material in work to follow, which may be of more legitimate concern to the psychopathologist. But to him, a book like the present raises somewhat forcibly an underlying question of ideational values. It is a case in which possible benefit to psychopathology or to society seems minute in proportion to the energy systematically and devotedly expended. Purposes of sublimation may have been well served; but not every one's mind endures, St. Antony-like, "ideationizing" so naked and unashamed. Of those more fortunate, there sang that great though here uncited philosopher of our author's tongue,

> "Wie wohl ist's dem, der dann und wann
> Sich etwas schönes dichten kann!"

LE SYMPATHIQUE ET LES SYSTEMES ASSOCIES: ANATOMIE CLINIQUE, SEMIOLOGIE ET PATHOLOGIE GENERALE DU SYSTEME NEURO-GLANDULAIRE DE LA VIE ORGANIQUE. Par A. G. Guillaume. Paper. Price, 6 francs 50 net. Pp. 160, with illustrations. Paris: Masson et Cie, 1920.

This little book of 160 pages deserves the attention of both the internist and the neurologist. It deals with the anatomy, physiology and pharmacology of the visceral nervous system and illustrates these fundamental subjects with simple and readily understandable diagrams. Such subjects as the

anatomy of the different sensory fibers of the visceral system and the distribution of the sympathetic fibers from the thoracico-lumbar cord to the spinal and cranial nerves and thence to the end organs are thoroughly dealt with.

The clinical chapters are brief and, beyond a few observations on the results of trauma occurring from war missiles, contain no new material.

Altogether the book is clearly written, well illustrated and complete from the point of view of anatomy and physiology. As a means of reviewing this subject, it is excellent.
